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VOLUME 22

1921

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

JUL 14 1921

VOLUME 22 NUMBER 1

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PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $5.00

Entered as Second-Class Matter, January 6, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Accepting for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

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American Journal of Diseases of Children

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THE DEGREE OF IMMUNITY TO DIPHTHERIA INSURED BY A NEGATIVE SCHICK TEST *

WILLIAM H. PARK, M.D.
NEW YORK

This communication was suggested by reading an article on an outbreak of tonsillitis suspected to be diphtheritic by Dr. S. A. Blauner.[1] Through the courtesy of Dr. Blauner, I was enabled to study the outbreak described. I saw the last three cases in the acute shape, and made cultures from the sick and convalescent children and from many of the healthy children distributed in the different parts of the institution. The laboratory and clinical material considered together lead me to somewhat different conclusions from those drawn by Dr. Blauner. Although the results of all the laboratory examinations were sent to him, he either failed to remember them or did not interpret them as I do.

Before considering this special outbreak, it seems desirable to state the evidence which supports the view that sufficient antitoxin in the body to prevent the development of a positive Schick test under ordinary conditions will prevent the development of diphtheria. There can be no doubt that the injection of 500 units of antitoxin will give children an immunity for a period of at least twelve days and usually twenty days. The proof of this is derived from its use in many outbreaks and in the exposed children of hundreds of thousands of families. The recent statement by Haven's that about 20 per cent. of diphtheria bacilli are of a different type from the one used to produce toxin for the immunization of horses, and that the available monovalent antitoxin will not completely neutralize the toxin made by this other type, and that, therefore, diphtheria may develop from infection with this other type in children showing a negative Schick test to the standard toxin, has not been borne out by experience nor confirmed by us in experimental work. The evidence that antitoxin is the only substance in the body that can prevent the toxin acting is also so conclusive that we can assume that a negative Schick test means the

* Received for publication, June 11, 1921.

1. Am. J. Dis. Child. **21**:472 (May) 1921.

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presence of sufficient antitoxin to neutralize it. The debatable points are: is the minimum amount of antitoxin required to prevent the Schick test sufficient to prevent the development of diphtheria and, if not, is it sufficient to prevent a toxemia. Closely bound up with the answer to these questions is the degree of certainty that the general practitioner can assume that, as ordinarily carried out, a negative Schick test proves the presence of at least this minimum amount of antitoxin. It will be wise to dispose of the last query first.

A Schick test requires the injection intracutaneously of one-fiftieth of a lethal dose for a 250 gm. guinea-pig in 0.1 c.c. or an equivalent amount of toxin in a different amount of fluid. We recommend one-fourtieth M. L. D. in 0.2 c.c. because we find the larger amount of fluid easier to give and, therefore, safer for the inexpert. Reports sent us some time ago of several series of cases with no positive Schick reactions caused us to obtain outfits from various manufacturers. Dr. Zingher found in a considerable number of instances that the available quantity of toxin in some of the outfits was much less than the required amounts. The children tested with these solutions gave negative Schick reactions when control tests made with standardized toxins gave positive ones. More recent tests of outfits from these firms have given uniformly good results.

Not only must the toxin be measured accurately, but the toxin must be delivered wholly intracutaneously. If injected a little too deep it will not be retained in the skin and will not exert the desired effect. If the small white area does not appear at the point of injection the Schick test cannot be relied on. Careful investigation leads us to believe that from 2 to 4 per cent. of the persons giving negative Schick tests do so because of error in technic. When great care has been taken to have the toxin strength correct and the injection given strictly intracutaneously, we have not discovered any errors. Too strong a toxin will give a larger percentage of positives than will the standard amount. The endeavor of Schick was to choose the amount that would as nearly as possible divide the susceptible from the immune.

During the past seven years my colleagues in the laboratory have made over 150,000 Schick tests, and have immunized about forty thousand of those giving a positive reaction. We have also tested from year to year about fifteen thousand children who are inmates in institutions so as to note any changes. There has been an average difference of about 2 per cent. in the annual readings. This we have considered as partly due to borderline cases and partly to errors in technic. The major part of this work has been directly carried out or has been supervised by Dr. A. Zingher and Dr. M. C. Schroder. We have during these years earnestly sought to discover all cases of

suspected diphtheria which occurred among those who had given negative Schick reactions, either before or after the toxin-antitoxin injections.

During this time we have learned of and investigated eighteen cases of suspected diphtheria in those who had given a negative Schick reaction. Two of these were in an institution in Brooklyn, two in an institution in New York, eight in Dr. Blauner's institution and six in their homes. In none of these cases has death occurred. Undoubtedly, there have been other cases concerning which information has not reached us. We are now cataloging 100,000 New York City school children who have given negative Schick tests, and 100,000 untested children from the same schools.

During the next year we are going to look up every case reported as diphtheria and determine the difference between the incidence of diphtheria in the two groups of children. In as far as possible, we will inquire into each case so as to have both the physician's original report and the results of the inspector's investigation. We hope by this method that within a year we may get a decision as to the degree of immunity from diphtheria insured to children giving a negative Schick reaction.

It is very difficult to decide whether any of the sixteen cases with positive cultures which had patches of pseudomembrane either confined to the tonsils or spreading to the pharyngeal borders were cases in which a faulty Schick test had been done, or whether they must be considered as moderate cases of diphtheria developing in spite of an amount of antitoxin sufficient to prevent a positive Schick test or whether they were due to other microbes, the diphtheria bacilli being present by accident and taking no active part. An important fact to remember is that whatever the final decision is as to the etiology of these cases that they are extremely rare.

A detailed discussion of the outbreak that occurred in Dr. Blauner's service will show the complexity of the factors involved. We will combine a consideration of the clinical observations supplied by Dr. Blauner and the laboratory findings of Miss Mann who carried out extensive investigations under the general direction of Dr. A. W. Williams and myself.

The clinical facts can be stated briefly as follows: In a ward of about twenty children ranging from 2 to 5 years of age, all but one of whom had given a negative Schick reaction, three cases of croupous tonsillitis developed December 23, one on the twenty-sixth, two on the twenty-eighth, one on the twenty-ninth and two on the thirty-first. Eight of these showed grayish patches on the tonsils. In five, the patches spread from the tonsils to the edges of the pharynx. One of the two cases developing on the thirty-first was mild and had no

exudate. The temperatures of the cases ranged from 102 to 104 F. on the first day, and dropped rapidly by lysis.

Dr. Blauner states in his article that at first he had no suspicion that the earliest three cases had diphtheria because of the previous negative Schick reactions, but when the positive laboratory reports came in he became convinced that they were cases of true diphtheria. The three cases I saw would have been considered by me as probably, but not at all certainly, diphtheria. The cultures from all the cases, except the ninth, which was very mild, contained virulent diphtheria bacilli. The mild case contained no diphtheria bacilli.

Four of the eight moderately severe cases had given negative Schick reactions on their entrance into the institution; three had become negative after injections, and one, though injected, had remained positive. The first seven [2] of the children received antitoxin and recovered promptly; the last two received no antitoxin and recovered equally rapidly. The possibilities seemed to be (1), that the amount of antitoxin as shown by the negative Schick reaction had been in these cases insufficient to prevent moderate diphtheria; (2) that some other infection had caused the croupous tonsillitis, and that the diphtheria bacilli were accidentally present in carriers, and (3) that the sore throat started by the pyogenic organisms had paved the way for the local action of the accompanying diphtheria bacilli. To test out these possibilities we omitted giving antitoxin in the last two cases which had just developed and one of which was very mild. These two cases progressed favorably as I felt certain they would and the children recovered as promptly as did those that had received antitoxin. This result suggested that whether or not the diphtheria bacilli had taken part in the formation of the exudate, the recovery was probably not due to the additional antitoxin injected.

A week later, January 7, cultures were made from twenty-seven of the children in the room in which the infected cases occurred, and from eleven of them virulent diphtheria bacilli were obtained. As these children had been given an immunizing injection of antitoxin, Dr. Blauner thought that the cessation of the cases, in spite of the presence of so many carriers, was a strong point in favor of the former amount of antitoxin having been insufficient to protect. This led me to make cultures from nineteen children taken at random from other parts of the building where no antitoxin immunization had been given. To our surprise, we found the carriers even more abundant here than in the ward from which the cases came. Miss Mann isolated diphtheria bacilli from the cultures made from ten of nineteen children (See accompanying table). All strains were tested and found to be toxin

2. There is some confusion as to whether it was the eighth case or an additional case that received no antitoxin. There is no doubt that there was a case with tonsillar exudate similar to the eighth case.

producers. The children in the general building were, therefore, apparently protected by the amount of antitoxin which had given a negative Schick reaction. With these results before us, we see that if the tonsillitis in the ward had been caused by some other infection than diphtheria, the throats, if not already infected, would almost necessarily have become carriers of the bacilli. Dr. Blauner's complete dependence on the culture results to solve the nature of the infection is not surprising, since health departments have so emphasized the value of the bacterial reports in suspected cases of diphtheria that many physicians believe that a positive report from a suspected case proves that the case is one of diphtheria.

RESULTS OF THE CULTURE EXAMINATION OF NINETEEN HEALTHY CHILDREN FROM WARDS IN WHICH NO DIPHTHERIA HAD OCCURRED

Name	Result	Name	Result
A. R.	Negative	L. E.	Diphtheria bacilli
A. M.	Diphtheria bacilli	L. M.	Diphtheria bacilli
B. F.	Diphtheria bacilli	M. L.	Diphtheria bacilli
B. A.	Diphtheria bacilli	R. L.	Negative
B. W.	Negative	S. L.	Negative
D. M.	Diphtheria bacilli	S. S.	Diphtheria bacilli
G. I.	Negative	W. S.	Negative
H. M.	Negative	W. J.	Diphtheria bacilli
H. D.	Diphtheria bacilli	Z. J.	Negative
H. M.	Negative		

The absence of bacilli in a culture properly made is fairly good proof that no bacilli are in the throat and, that, therefore, the case is not one of diphtheria. If bacilli are present, the probability of diphtheria being present is greater or less according to the more or less characteristic appearance of the lesions. The laboratory indicates the presence or absence of the bacilli and the danger of the case spreading infection; the clinical examination determines the probability or certainty of the presence of diphtheria. It would be more helpful if the laboratory could make the diagnosis, but, unfortunately, it cannot. If we apply the above facts to Dr. Blauner's cases, we see that as certainly half of the children in the institution were carrying virulent diphtheria bacilli in their throats during the month of January, that whatever nasal or throat infection took place diphtheria bacilli would probably be associated with it. With 50 per cent. of the normal cases carrying bacilli, practically every diseased child would almost immediately become a carrier.

CONCLUSIONS

A negative Schick test in cases which have an active immunity, either natural or acquired, when the toxin used and technic employed have been suitable, gives an almost complete security from diphtheritic disease, not only for the immediate time but for the future.

Those that have natural antitoxin and those who acquire it through toxin-antitoxin injections may harbor diphtheria bacilli, and if they later suffer from tonsillitis, due to other microbes, throat cultures will contain diphtheria bacilli. The positive culture alone suggests but does not establish that the suspected case is one of diphtheria.

Under unusual conditions it is possible that when diphtheria bacilli are present in a throat which becomes the seat of some other infection they may develop their toxin and cause superficial lesions in the mucous membrane even though the cases have sufficient antitoxin to give a negative Schick reaction. Cases which present this possibility are rare, and when met with by us have done well without additional injection of antitoxin.

In the outbreak discussed in this communication, the fact that while an extraordinarily large percentage of all the children in the institution were carriers of virulent bacilli, only one group developed cases of tonsillitis with pseudomembranous patches makes it uncertain that the cases were diphtheria.

THE RANGE AND DISTRIBUTION OF BLOOD PRESSURES IN NORMAL CHILDREN

CLINICAL APPLICATION OF STATISTICAL METHODS TO THE INTERPRETATION OF DEVIATION FROM THE NORMAL AVERAGE *

HAROLD K. FABER M.D., AND CHARLES A. JAMES, M.D.
SAN FRANCISCO

The usefulness of blood pressure determination as means of diagnosis has long suffered from the lack of suitable standards which would define accurately the limits of normal tension and of pathologic hypotension or hypertension. We are abundantly supplied with averages, but averages have, at best, an academic value unless they are supplemented by standards of normal deviation from the average. Particularly in pediatrics, the lack of such standards of deviation has resulted in the neglect of sphygmomanometry as having too little practical value. It has seemed to us that much useful information regarding the cardiovascular functions and disturbances of childhood might be obtainable once the desired normal standards were established, and it was in an effort provisionally to formulate such standards that the present study was undertaken.

Not only blood pressure statistics but nearly all medical statistics have been organized in such a way as to give averages only. In some instances, authors have attempted to furnish maximum and minimum values also, but, realizing the unreliability of absolute maximum and minimum findings, they have determined the average maximum and average minimum values, a method which may justly be criticized. In general, the range has been disregarded, not because it is not important to know but because it is very difficult satisfactorily to define.

GENERAL PRINCIPLES [1]

It has of course been realized that individual measurements, as of blood pressure, always deviate to some extent from the average and

* Received for publication Dec. 17, 1920.

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1. For a discussion of the principles and application of the statistical methods we have employed, see Yule,[2] Davenport [3] or Rugg.[4]

2. Yule, G. U.: An Introduction to the Theory of Statistics, Griffin & Co., 1912.

3. Davenport, C. B.: Statistical Methods with Special Reference to Biological Variation, New York, John Wiley & Sons, Inc., 1914.

4. Rugg, H. O.: Statistical Methods Applied to Education, Houghton Mifflin Co., 1917.

that when we compare such individual measurements with the average in order to decide whether or not abnormality is present we must make allowances for normal deviation. Hence we are accustomed in practice to speak of a "normal range," within the limits of which all measurements must be regarded as normal, but it is noteworthy that no two clinicians can be found to agree on the exact limits. Nevertheless there is no difficulty, even on inspection, in classifying as normal those readings which are near the average, or as abnormal those which show extreme deviation from the average. Clearly, then, there is an intermediate zone in the range of pressures, between the certainly normal and the certainly abnormal where interpretation is in doubt and where our chief difficulties lie. In this zone, how are we to measure deviations in such a way as to discover their true significance? Kilgore [5] in his illuminating paper, has recently called attention to the potential usefulness of the statistical method of frequency distribution for the solution of medical problems of this nature, and it is by this method that we have organized our material. An outline of the method will serve to make its applications clearer.

THE METHOD OF FREQUENCY DISTRIBUTION

If we take the blood pressures of a large series of presumptively normal individuals, we shall find that a majority occur at or near the average pressure of the whole group, and that at levels of pressure more distant from the average a smaller number of cases occur. If the distribution of such a series were plotted, the curve would approximate in form the mathematical probability or frequency curve, shown in Figure 1. The latter expresses not only the ideal distribution of a homogeneous series of normal data, but it can also be used to show the proportion of cases which will show any given deviation from the average, and also the chances that a given individual will show a given deviation from the average. Similarly, by clinical inference, it is clear that the nearer his pressure is to the average of all normal individuals, the greater are the chances that his pressure is normal; and, conversely, the greater his deviation from the average, the greater the chances that his pressure is abnormal, and the smaller the chances that his pressure is normal. In the mathematical statistical system the proportions and chances referred to are determined by the use of a measure of deviation from the mean, or average, which is known as the standard deviation of Pearson.[6] By dividing the devia-

5. Kilgore, E. S.: Relation of Quantitative Methods to the Advance of Medical Science, J. A. M. A. **75**:86 (July 10) 1920.

6. The standard deviation is the square root of the arithmetic mean of the square of the deviations from the average of the distribution. It is designated by the Greek letter sigma (σ).

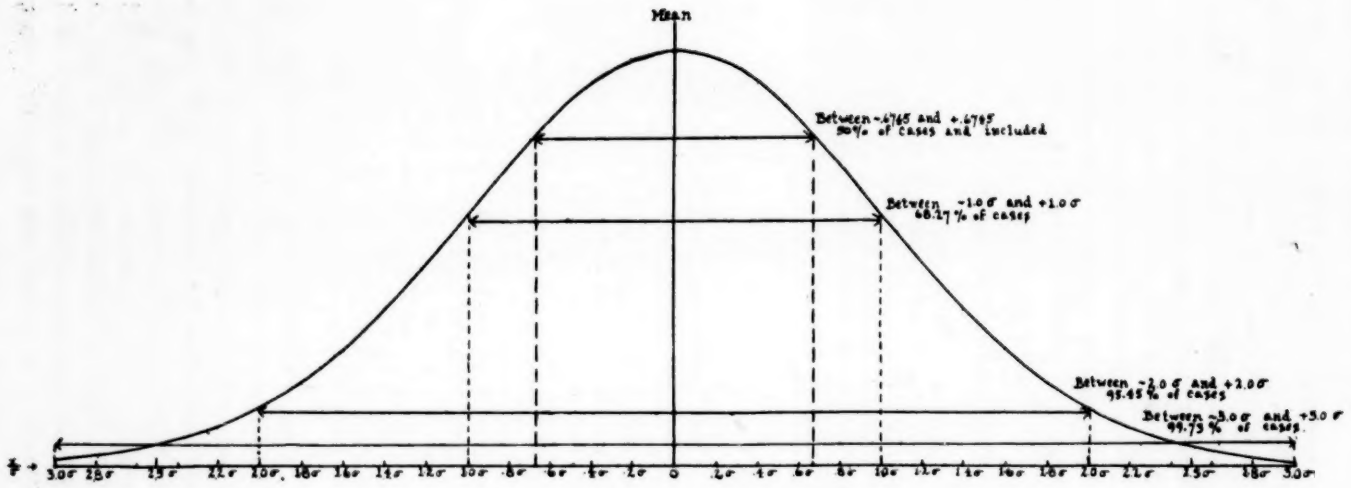
Mean
Between -.6745 and +.6745
50% of cases and included
Between -1.0 σ and +1.0 σ
68.27 % of cases
Between -2.0 σ and +2.0 σ
95.45 % of cases
Between -3.0 σ and +3.0 σ
99.73 % of cases
3.0σ 2.8σ 2.6σ 2.4σ 2.2σ 2.0σ 1.8σ 1.6σ 1.4σ 1.2σ 1.0σ .8σ .6σ .4σ .2σ 0 .2σ .4σ .6σ .8σ 1.0σ 1.2σ 1.4σ 1.6σ 1.8σ 2.0σ 2.2σ 2.4σ 2.6σ 2.8σ 3.0σ

Fig. 1.—Normal frequency curve.

tion of a given measurement from the average by the standard deviation, we obtain a quotient, to which we have applied the term "deviation index," having a definite relation to the normal, or ideal distribution, and showing the normal proportion of cases occurring at the given level. The deviation index would appear to have an important clinical value in defining the significance of individual variations from the average, and also in determining the normal and abnormal range. In order to obtain it in any given case, it is necessary only to know the corresponding average and standard deviation. The exact meaning of the deviation index is illustrated by the following tabulations:

In a series of normal cases ideally distributed,

49.86	per cent.	will have a	deviation	index	between	−3.0[7]	and	0
47.7	per cent.	will have a	deviation	index	between	−2.0	and	0
34.1	per cent.	will have a	deviation	index	between	−1.0	and	0
34.1	per cent.	will have a	deviation	index	between	+1.0	and	0
47.1	per cent.	will have a	deviation	index	between	+2.0	and	0
49.86	per cent.	will have a	deviation	index	between	+3.0	and	0

and, conversely,

0.14	per cent	will have a	deviation	index of	less than	−3.0
2.3	per cent.	will have a	deviation	index of	less than	−2.0
34.1	per cent.	will have a	deviation	index of	less than	−1.0
50.0	per cent.	will have a	deviation	index of	less than	0
50.0	per cent.	will have a	deviation	index of	more than	0
34.1	per cent.	will have a	deviation	index of	more than	+1.0
2.3	per cent.	will have a	deviation	index of	more than	+2.0
0.14	per cent.	will have a	deviation	index of	more than	+3.0

hence, the chances that the index of a normal individual will be

less than	−3.0	are 1	in	741	or 1	to	740
less than	−2.0	are 1	in	44	or 1	to	43
less than	−1.0	are 1	in	6.3	or 1	to	5.3
less than	0	are 1	in	2	or 1	to	1
more than	0	are 1	in	2	or 1	to	1
more than	+1.0	are 1	in	6.3	or 1	to	5.3
more than	+2.0	are 1	in	44	or 1	to	43
more than	+3.0	are 1	in	741	or 1	to	740[8]

The deviation index, therefore, may be converted into "betting odds," expressing the chances that any normal person, selected at random, will be found to have a pressure deviating from the average as much as, or more than, that of the patient under examination. If, for instance, a patient has a systolic pressure of 80 mm. and the deviation index of 80 mm. is −2, we compare this pressure with the odds of 1 in 44, or 1 to 43, that any normal individual of the same age and sex will be found to have a pressure of 80 mm. or less. In the language of the race track, this is a "long shot" but one with a chance to win—

7. The − sign signifies deviation below the average, and the + sign, deviation above the average.

8. Table 1 gives more complete figures.

the patient may be normal. When the odds mount to 1 to 740, as they do at D.I. ± 3, the chances of "winning"—of normality—become small indeed. For practical purposes, they will, we think, be granted to amount to a certainty of "loss"—the individual cannot be regarded as normal—the limits of the normal range have been passed.

THE RANGE

We may, therefore, make certain deductions from the frequency method as to probable normal range. The result, however, is not one

TABLE 1

A D. I.	B Per Cent.	C Per Cent.	D Chances	E Chances
±0.0	0.0	50.0	1 in 2	1 to 1
0.1	4.0	46.0	2.2	1.2
0.2	7.9	42.1	2.4	1.4
0.3	11.8	38.2	2.6	1.6
0.4	15.5	34.5	2.9	1.9
0.5	19.2	30.8	3.2	2.2
0.6	22.6	27.4	3.7	2.7
0.7	25.8	24.2	4.1	3.1
0.8	28.8	21.2	4.5	3.5
0.9	31.6	18.4	5.4	4.4
1.0	34.1	15.9	6.3	5.3
1.1	36.4	15.6	7.3	6.3
1.2	38.5	11.5	8.7	7.7
1.3	40.3	9.7	10	9.3
1.4	41.9	8.1	12	11
1.5	43.3	6.7	15	14
1.6	44.5	5.5	18	17
1.7	45.5	4.5	22	21
1.8	46.4	3.6	28	27
1.9	47.1	2.9	35	34
2.0	47.7	2.3	44	43
2.1	48.2	1.8	56	55
2.2	48.6	1.4	72	71
2.3	48.9	1.1	93	92
2.4	49.2	0.8	122	121
2.5	49.4	0.6	162	161
2.6	49.5	0.5	213	212
2.7	49.6	0.4	286	285
2.8	49.7	0.3	278	277
2.9	49.8	0.2	527	526
3.0	49.86	0.14	741	740
3.1	49.90	0.10	1,030	1,030
3.2	49.93	0.07	1,450	1,430
3.3	49.95	0.05	2,070	2,070
3.4	49.97	0.03	2,940	2,940
3.5	49.98	0.02	4,270	4,270
4.0	49.997	0.003	31,550	31,550
5.0	49.99997	0.00003	3,448,280	3,448,280

Column B: percentage of cases normally found between D. I. and mean, or average (D. I. = 0).
Column C: percentage of cases showing a greater deviation from mean in the same direction.
Column D: chances that a normal individual will show the same or a greater deviation from mean in the same direction.
Column E: chances for to chances against, a normal individual deviating more from mean; i. e., having larger D. I.

absolute range, within which every measurement is normal and without which everything is abnormal, but an arrangement much more like the actual subdivision of cases made roughly by all clinicians. By means of the deviation index, we can, perhaps, state fairly that deviations of less than 1 are unquestionably normal, while deviations of more than 3 are almost certainly abnormal, but between these two limits there is a

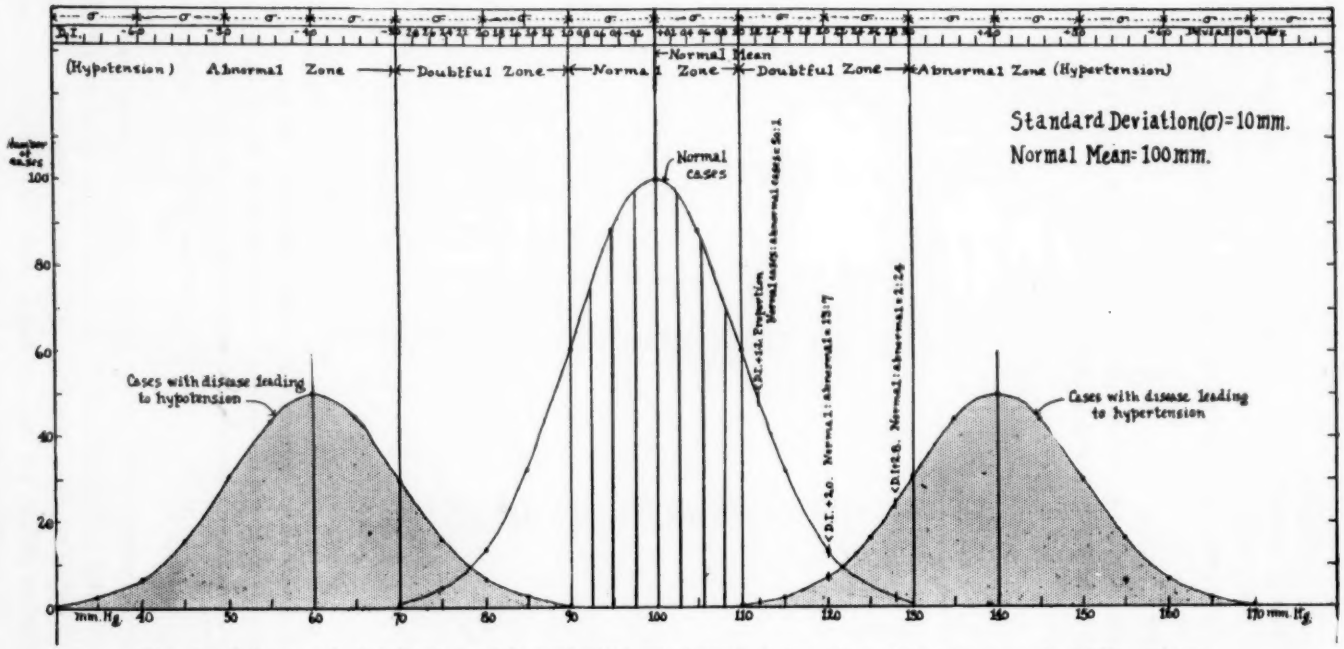

Fig. 2.—Scheme of distribution of hypothetica¹ unselected series of systolic pressures in 1,200 subjects.

doubtful zone, where measurements may occur in either normal or pathological cases. The reason for this will be made clear by consulting Figure 2, showing schematically the distribution of pressures in a single unselected series of patients with and without disease affecting vascular tension.

Within the doubtful zone it will be seen that both the normal and pathologic cases are represented, but it will be noted also, that as the distance from the normal average increases, the number of normal cases decreases, and the number of abnormal cases increases. It will,

TABLE 2.—DISTRIBUTION OF SYSTOLIC PRESSURE

Mm.	Number of Cases	Mm.	Number of Cases
75-79	1	105-109	73
80-84	4	110-115	34
85-89	14	115-119	4
90-94	20	120-124	7
95-99	91	125-129	1
100-104	160		

Average, 102.5 mm. Standard deviation, 6.76 mm.

TABLE 3.—IDEAL DISTRIBUTION OF CASES ENUMERATED IN TABLE 2

Mm.	Actual Number	Ideal Number	Deviation* Mm.	Deviation Index (D.I.)*
75-79	1	0	—25	—3.7
80-84	4	2	—20	—2.96
85-89	14	10	—15	—2.22
90-94	20	41	—10	—1.48
95-99	91	93	— 5	—0.74
100-104	160	118	0	0
105-109	73	93	+ 5	+0.74
110-114	34	41	+10	+1.48
115-119	4	10	+15	+2.22
120-124	7	2	+20	+2.96
125-129	1	0	+25	+3.7

* Of the midpoint of each interval.

therefore, be seen that the deviation index, measuring the probabilities as they increase or decrease with the distance from the central average, is the true measure by which to estimate the significance of any observation, and the only measure by which observations in the doubtful zone can be evaluated.

ORGANIZATION OF ACTUAL STATISTICS

Actual series of clinical observations, unless they be very large, or of a nature which makes it extraordinarily simple to exclude abnormal data, rarely follow exactly the ideal frequency distribution. It is, however, possible to compute with fair approach to the absolute values

both the standard deviation and the mean, or average, from a relatively small number of observations. The degree of inaccuracy can also be computed. Once the standard deviation and mean have been found, the ideal distribution can be calculated and the range limits, above discussed, determined. It will, perhaps, assist the reader to give an example which will illustrate the principles involved, the method by which our final tables were calculated and the method by which they can be applied practically.

In the group of boys between 9 and 11 years, the 409 observations of systolic pressure were distributed as shown in Table 2.

Applying the standard deviation as the controlling factor in frequency distributions, we can determine approximately the ideal distribution of these cases as shown in Table 3.

The curves for the actual and theoretical distribution are shown in Figure 3.

Taking the standard deviation (D.I. = 1) as the range of absolute normal, and three times the standard deviation (D.I. = 3) as the inner limit of the absolutely abnormal, the three ranges would be:

Normal: 102.5 mm. ± 6.76 mm. = 95.74 mm. to 109.26 mm., or 96 — 109 mm.

Doubtful: 102.5 mm. ± 6.76 to 102.5 mm. ± 3 × 6.76 = 82.3 — 95.7 and 109.3 — 122.8 mm., or 82 — 96 mm. and 109 — 123 mm.

Abnormal: 102.5 mm. ± < 3 × 6.76 mm. = under 82.3, over 122.8 mm., or under 82 mm. and over 123 mm.

METHODS OF APPLICATION

Let us now suppose that we have examined a boy aged 10, and have found his systolic pressure to be 125 mm. The average being 102.5 mm. for boys of this age, his deviation is [9]

$$125 - 102.5 = + 22.5 \text{ mm.}$$

Since the standard deviation has been found to be 6.76 mm., the deviation index is

$$\frac{+22.5}{6.76} = + 3.33$$

Reference to Table 1 will show (a) that out of 1,000 normal cases more than 999 will have a smaller index; (b) that less than 0.1 per cent. of normal cases have an index as large; (c) that the chances that a given individual, if normal, will have an index as large are less than 1 to 2,070. By the definitions of the three zones, we note that an index of 3 or over places the individual in the abnormal zone. Hence, we can state that this individual is suffering from pathologic hypertension.

9. The sign indicating whether the pressure is above or below the average is kept throughout.

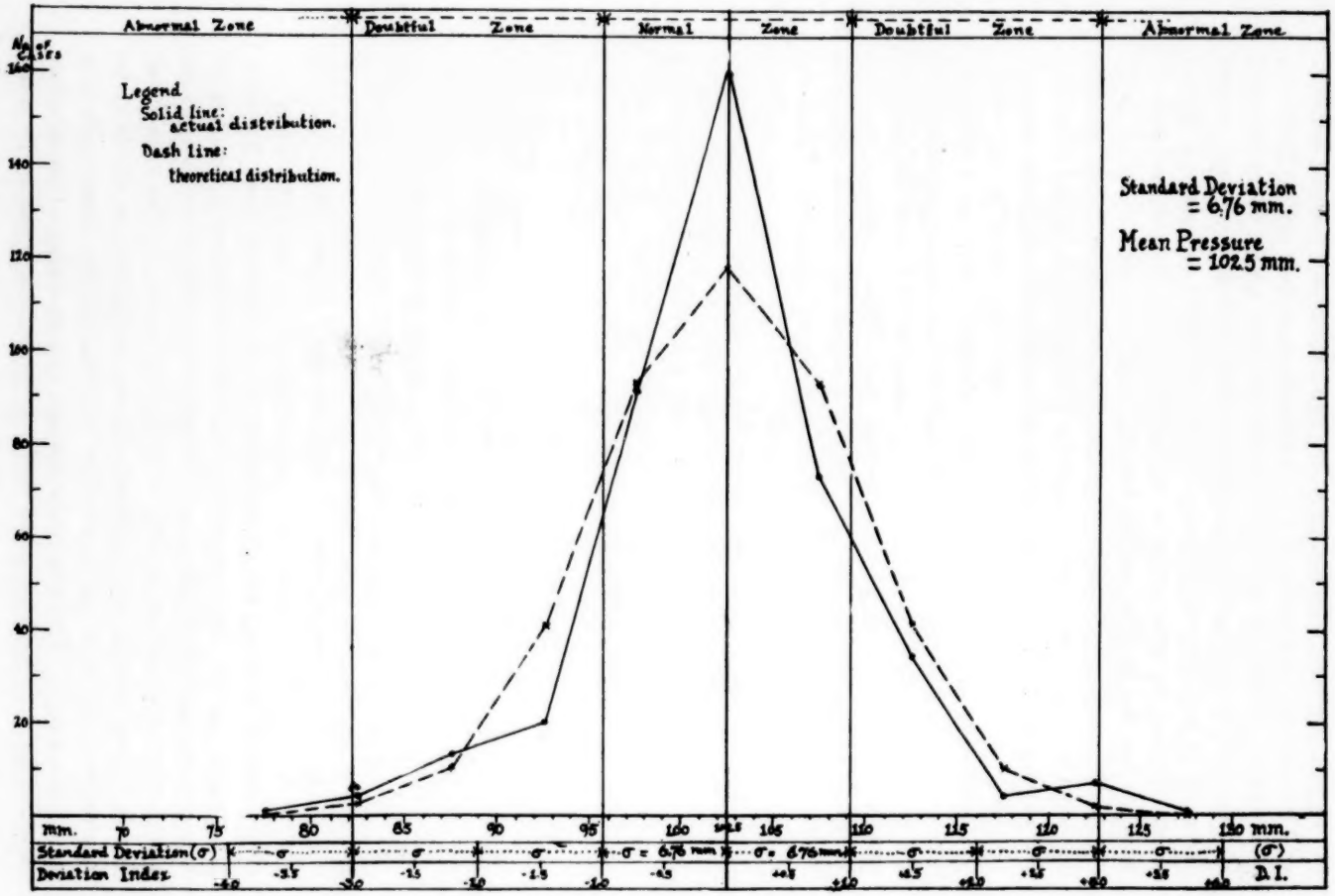

Fig. 3.—Actual and theoretical distribution of systolic pressures in the group of boys between 9 and 11 years of age.

Again, apply the method to a boy aged 10 with a systolic pressure of 116 mm. The deviation of + 13.5 mm. gives an index of + 2, placing the reading between 1 and 3, or in the doubtful zone. Since only 2.3 per cent. of normal cases have an index less than this, we may regard the case as decidedly suspicious, but not definitely pathological, unless so determined by other tests or symptoms. Such a case would, however, be one for further careful investigation.

Another patient of the same age and sex with a pressure of 96 mm., having a deviation of — 6.5 mm. and a D.I. of — 0.97 would be within the normal limit of 1 and without further consideration be regarded as normal.

While the accurate evaluation of "chances" requires a table of values (Table 1), one learns, after applying the method a few times, the relative significance of the various values for D.I., and for practical purposes can often dispense with the table. One thinks of a given pressure as a + 2.5 pressure, or a — 3 pressure, or a + 0.4 pressure, these terms having come to assume much more significance than the mere level of the mercury column alone.

It need hardly be said that the method is applicable to diastolic and pulse pressure as well as to systolic pressure. It is also applicable to pulse rates, and to the pulse rate—pulse pressure product which has been considerably used as an index of cardiac output. For all these we have constructed tables for clinical use.

DESCRIPTION OF MATERIAL

The observations on which the present study is based were made personally by us (to keep the personal factor as constant as possible) on 651 boys and 450 girls (1,101 individuals), and comprise a total of 2,710 measurements of systolic and diastolic pressures. Large as is the number, the necessity of grouping the data into age classes, as shown in Table 4, with different means and deviations, has reduced the reliability of our conclusions. The reliability of means increases as the square root of the number of variates; that of the standard deviations as the square root of twice the number of variates. It was, therefore, recognized that in order materially to increase the approximation of our results to the absolute, would require an enormous increase in the number of observations, a task beyond our powers, or the material at hand. Some idea of the accuracy of our means can be obtained by comparison with the figures of other writers, notably those of Judson and Nicholson.[10] Unfortunately for us, the readings of most other workers have either been taken under different conditions or by other

10. Judson, C. F. and Nicholson, P.: Blood Pressure in Normal Children, Am. J. Dis. Child. **8**:257 (Sept.) 1914.

methods. Judson and Nicholson, using a modified Erlanger instrument (checking the auscultatory method), and examining children lying down, found an average systolic pressure of 91.6 at age 4, for both sexes, and of 106.1 at age 14. Katzenberger,[11] using the auscultatory method obtained values of 93 at age 4 and 119 at age 14. Our values for the two sexes are 88.9 at age 4 and 109.6 at age 14. In diastolic pressure our readings do not corrersponds closely with those of Judson and Nicholson, who failed to find any constant variation with age, though their figures show considerable irrregular fluctuation. It is possible that the difference in position may account for the discrepancy between our figures and theirs. Katzenberger found, as we did, a progressive rise in diastolic pressure, but the figures themselves are not comparable because he used the fifth phase as the diastolic point. Differences in method, therefore, make it impossible to combine the various collections of data in order to increase the accuracy of the results.

TABLE 4.—DISTRIBUTION OF DATA

Age	Number of Individuals		Number of Observations	
	Boys	Girls	Boys	Girls
3	12	7	22	16
4	23	19	29	50
5	27	14	57	29
6	51	24	108	54
7	50	32	102	72
8	46	45	113	128
9	68	57	144	155
10	47	43	121	109
11	60	47	144	118
12	54	53	136	140
13	79	37	174	87
14	42	32	125	82
15	49	17	172	45
16	30	18	87	49
17	13	5	36	6
Total	651	450	1,570	1,140
Total both sexes	1,101		2,710	

In examining our children, cards were made out showing age to the nearest birthday, sex, weight, height, sitting height, pulse, systolic and diastolic pressures; date, time of day; remarks on diagnosis or anything unusual in the history or physical examination. The majority of readings were taken between 9:30 a. m. and noon. A certain number of cases were excluded from our normal series on suspicion of abnormality in physical condition. An effort was made to take three consecutive readings of blood pressure and, simultaneously, of pulse, but this was not done in every case.

The auscultatory method was used with a cuff suited in width to the arm. About 90 per cent. of the measurements were taken with a

11. Katzenberger, A.: Puls und Blutdruck bei gesunden Kindern, Ztschr. f. Kinderh. **9**:167, 1913.

10 cm. cuff, and the remainder either with a 7.5 cm. cuff for the smallest children, or with the adult 12.5 cm. cuff for the largest. The stethoscope was applied below the cuff, as lightly as possible. All readings

TABLE 5.—Systolic Pressures

Age		1 Mean	2 4 P. E.	3 σ	4 4 P. E.	5 Number
3-4-5	Boys	89.4	1.4	5.32	0.96	107
	Girls	88.5	1.8	6.54	1.24	96
4-5-6	Boys	92.8	1.2	6.20	0.84	192
	Girls	90.7	1.5	6.24	1.04	133
5-6-7	Boys	94.5	1.1	6.38	0.76	263
	Girls	93.8	1.6	7.30	1.12	152
6-7-8	Boys	97.3	1.0	6.65	0.68	321
	Girls	97.3	1.2	7.36	0.88	249
7-8-9	Boys	99.2	0.9	6.54	0.68	354
	Girls	100.2	1.0	7.06	0.68	352
8-9-10	Boys	100.9	0.8	6.02	0.56	373
	Girls	101.2	0.9	6.79	0.68	387
9-10-11	Boys	102.4	0.9	6.64	0.64	407
	Girls	102.6	1.0	7.06	0.68	380
10-11-12	Boys	104.1	0.9	6.61	0.64	402
	Girls	104.3	1.1	7.64	0.76	366
11-12-13	Boys	105.9	0.8	6.73	0.60	456
	Girls	106.2	1.0	7.03	0.68	342
12-13-14	Boys	107.6	0.8	6.02	0.56	426
	Girls	108.4	1.0	6.17	0.68	305
13-14-15	Boys	110.1	0.8	6.72	0.60	467
	Girls	109.5	1.2	6.31	0.84	209
14-15-16	Boys	112.2	1.0	7.05	0.76	375
	Girls	112.7	1.4	6.79	1.00	171
15-16-17	Boys	115.3	1.1	7.00	0.76	292
	Girls	114.3	2.1	7.82	1.48	102

"4 P. E." (4 times the probable error), Column 2, indicates the limits of reliability for the mean; and 4 P. E., Column 4, for the standard deviation. The true mean or the true standard deviation should occur within 4 P. E. of the found values, shown in Columns 1 and 3. Column 5 gives the number of observations included in the final calculations after elimination of extreme variates.

TABLE 6.—Diastolic Pressures

Age		Mean	4 P. E.	σ	4 P. E.	Number
3-4-5	Boys	60.3	1.8	6.75	1.24	108
	Girls	59.8	1.9	6.70	1.32	93
4-5-6	Boys	62.7	1.5	7.85	1.08	194
	Girls	62.3	1.4	6.05	1.00	129
5-6-7	Boys	64.0	1.3	7.70	0.92	263
	Girls	64.1	1.7	7.50	1.16	153
6-7-8	Boys	65.4	1.1	7.27	0.76	319
	Girls	66.0	1.3	7.57	0.84	251
7-8-9	Boys	66.7	1.0	7.00	0.72	356
	Girls	67.9	1.0	7.29	0.76	353
8-9-10	Boys	67.8	1.0	6.89	0.68	379
	Girls	69.4	0.9	6.45	0.64	389
9-10-11	Boys	68.9	0.8	5.88	0.56	409
	Girls	70.3	0.9	6.62	0.64	379
10-11-12	Boys	69.9	0.7	5.12	0.48	397
	Girls	71.4	0.9	6.53	0.68	364
11-12-13	Boys	71.1	0.6	4.82	0.44	449
	Girls	71.9	1.0	6.80	0.72	342
12-13-14	Boys	71.9	0.6	4.90	0.44	431
	Girls	73.0	1.0	7.15	0.72	306
13-14-15	Boys	73.3	0.6	4.78	0.44	465
	Girls	73.5	1.6	8.50	1.12	213
14-15-16	Boys	74.6	0.8	5.94	0.56	383
	Girls	76.4	2.0	10.00	1.44	175
15-16-17	Boys	75.8	1.0	6.18	0.68	294
	Girls	79.1	2.6	9.90	1.84	105

were made with the mercury falling. All patients were in the sitting position with the arm horizontal, resting on a table. A brief preliminary rest was given. If the first measurement showed a striking dis-

crepancy from the next two, a fourth was taken and the first discarded. This was done because the excitement of the first application of the cuff, sometimes raised the pressure considerably. As a rule, this rapidly

TABLE 7.—PULSE PRESSURES

Age		Mean	4 P. E.	σ	4 P. E.	Number
3-4-5	Boys	29.6	1.8	6.75	1.24	108
	Girls	29.6	1.7	6.08	1.20	94
4-5-6	Boys	30.6	1.3	6.45	0.88	193
	Girls	29.6	1.5	6.25	1.04	131
5-6-7	Boys	31.1	1.0	5.60	0.68	264
	Girls	29.8	1.5	6.65	1.04	151
6-7-8	Boys	32.0	0.9	6.00	0.64	322
	Girls	31.7	0.9	5.14	0.60	250
7-8-9	Boys	33.1	0.8	5.73	0.60	353
	Girls	32.4	0.8	5.17	0.52	346
8-9-10	Boys	33.8	0.8	5.59	0.56	371
	Girls	32.1	0.7	5.21	0.52	388
9-10-11	Boys	33.9	0.8	5.72	0.56	406
	Girls	32.3	1.0	6.78	0.68	375
10-11-12	Boys	34.6	0.8	6.20	0.60	397
	Girls	32.9	0.9	6.57	0.68	359
11-12-13	Boys	35.2	0.8	6.34	0.56	450
	Girls	33.6	1.0	6.89	0.72	338
12-13-14	Boys	36.1	0.8	6.23	0.56	434
	Girls	35.2	1.0	6.46	0.72	302
13-14-15	Boys	37.5	0.7	5.97	0.53	470
	Girls	35.3	1.5	7.32	0.96	207
14-15-16	Boys	38.5	0.8	6.07	0.60	384
	Girls	36.1	1.7	8.38	1.20	174
15-16-17	Boys	39.6	1.0	6.43	0.71	294
	Girls	34.7	1.8	6.91	1.28	106

TABLE 8.—PULSE RATES

Age		Mean	4 P. E.	σ	4 P. E.	Number
3-4-5	Boys	98.8	2.6	9.50	1.80	100
	Girls	100.7	3.7	12.60	2.60	85
4-5-6	Boys	94.5	2.1	10.92	1.48	194
	Girls	98.0	2.9	11.66	2.04	119
5-6-7	Boys	92.0	2.0	11.80	1.36	267
	Girls	96.4	2.6	12.21	2.00	132
6-7-8	Boys	88.5	1.8	11.98	1.28	323
	Girls	95.6	2.0	10.28	1.44	187
7-8-9	Boys	86.4	1.6	10.91	1.08	358
	Girls	91.7	1.6	10.25	1.12	308
8-9-10	Boys	86.6	1.3	9.31	0.92	376
	Girls	91.4	1.6	11.10	1.16	343
9-10-11	Boys	86.3	1.2	9.35	0.88	407
	Girls	92.0	1.6	10.62	10.8	346
10-11-12	Boys	85.3	1.2	9.07	0.88	401
	Girls	93.1	1.7	11.53	1.24	325
11-12-13	Boys	85.1	1.2	9.20	0.80	457
	Girls	92.7	1.7	11.26	1.24	305
12-13-14	Boys	83.7	1.3	9.95	0.92	436
	Girls	90.9	1.8	10.76	1.24	270
13-14-15	Boys	82.8	1.3	10.15	0.88	472
	Girls	89.1	2.1	10.48	1.48	182
14-15-16	Boys	81.4	1.3	9.58	0.92	384
	Girls	88.2	2.2	10.09	1.56	153
15-16-17	Boys	81.4	1.5	9.55	1.08	295
	Girls	89.6	3.5	12.20	2.48	88

The necessity of counting pulse over 15 second periods and multiplying by four has introduced a technical difficulty in class interval distribution, while multiplying the observational error, which is probably considerable when pulse is taken by palpation in children. Our pulse rate values are, therefore, approximate only, both for the mean and for the standard deviation.

subsided and the children were not further disturbed by the test. Systolic pressure was taken as the point at which the first clear, continuous sounds appeared; the diastolic, at the fourth phase. The pulse

was taken by an assistant. The phases are nearly always distinctly marked after the third year, but they are often better elicited after the arm is raised and the hand clenched and unclenched a few times. Most of the children were school children, but about 150 were patients in the Children's Clinic suffering from minor complaints who appeared to be for practical purposes normal.

In compiling our statistics, frequency distributions were tabulated for age groups of three years [12] using class intervals of 5 mm. of mercury and the mean and standard deviation computed from each distribution was assigned to the middle year. Thus, the group 3-4-5 was taken as the type of year 4; 4-5-6, of year 5, and so on. This method was adopted when it was found that the number of measures in each

TABLE 9.—PULSE PRESSURE—PULSE PRODUCT

Age		Mean	4 P. E.	σ	4 P. E.	Number
3-4-5	Boys	2880	180	653	128	100
	Girls	2880	210	700	148	84
4-5-6	Boys	2830	130	671	92	194
	Boys	2850	170	679	120	118
5-6-7	Boys	2800	100	624	72	267
	Girls	2680	140	585	100	127
6-7-8	Boys	2820	100	645	68	323
	Girls	2800	120	634	84	205
7-8-9	Boys	2850	90	618	64	360
	Girls	2900	100	625	68	308
8-9-10	Boys	2870	90	648	56	377
	Girls	2890	90	642	68	343
9-10-11	Boys	2870	80	578	56	409
	Girls	2920	110	731	76	346
10-11-12	Boys	2880	80	550	52	401
	Girls	3000	120	786	84	325
11-12-13	Boys	2970	80	643	56	454
	Girls	3160	130	821	88	305
12-13-14	Boys	3010	90	707	64	436
	Girls	3200	130	781	92	270
13-14-15	Boys	3070	80	669	60	472
	Girls	3150	160	800	116	182
14-15-16	Boys	3100	90	643	64	385
	Girls	3120	170	781	120	153
15-16-17	Boys	3070	90	593	68	296
	Girls	3010	190	664	136	88

group was too small to give us a smooth curve. Use was then made of the statistical method of smoothing in which the average of three successive, related measures, a, b, c, is taken as the true b; thus,

$$b = \frac{a+b+c}{3} \quad \text{(Rugg,[4] p. 184).}$$

Since we have wished to make the practical use of the tables as simple as possible we have also made a further set of figures which we

12. We disagree with Wolfensen-Kriss [13] who states that blood pressure varies with height and weight rather than with age. We have plotted our systolic pressures against both height and weight and sitting height and have found that the spread of distribution is just as great for any of these as it is for age. Age appears to be, as a whole, as accurate (and perhaps as fallacious) a measure of development as weight or one of the linear measurements.

13. Wolfensen-Kriss, P.: Ueber den Blutdruck im Kindesalter, Arch. f. Kinderh. **53**:332, 1910.

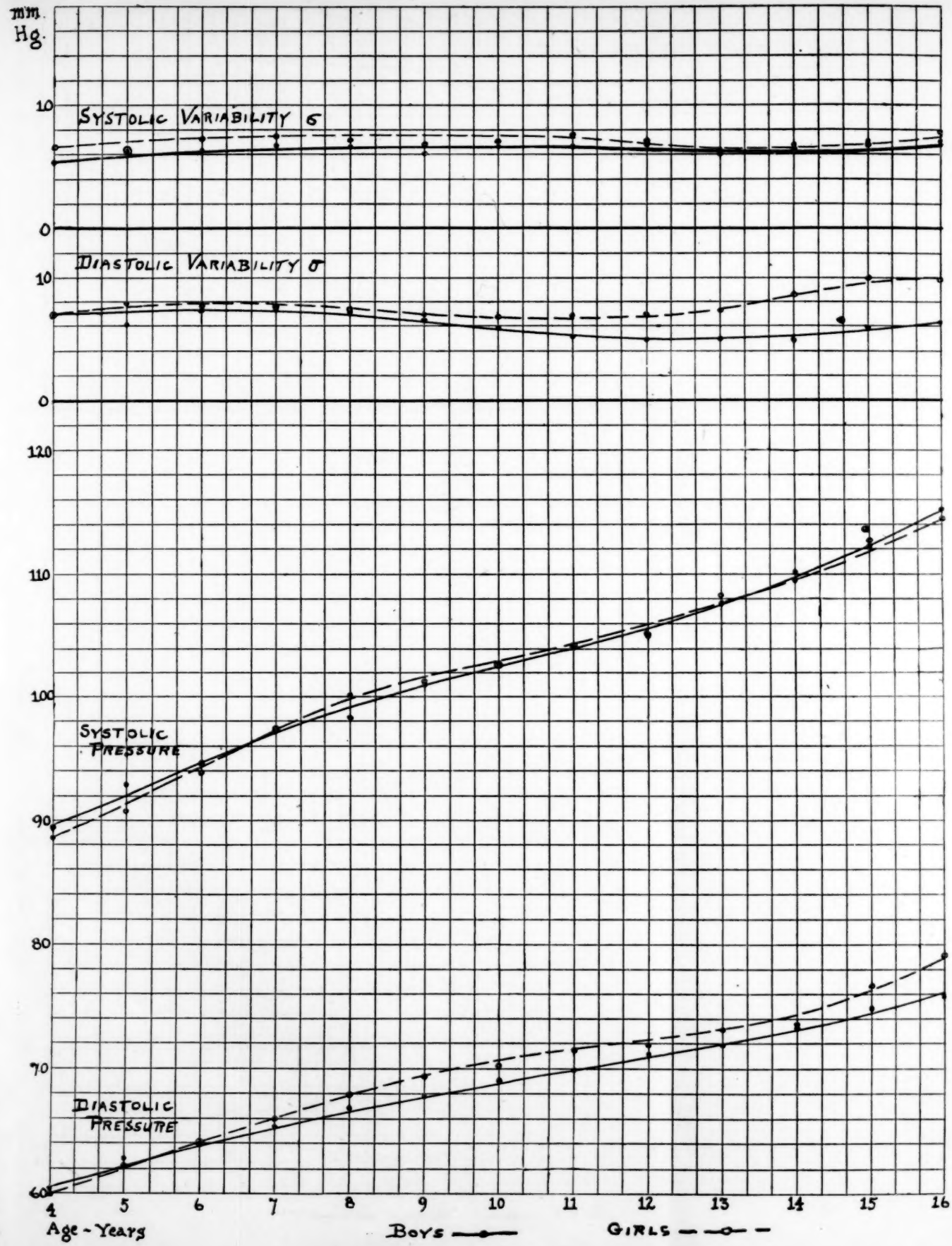

Fig. 4.—Systolic and diastolic pressures.

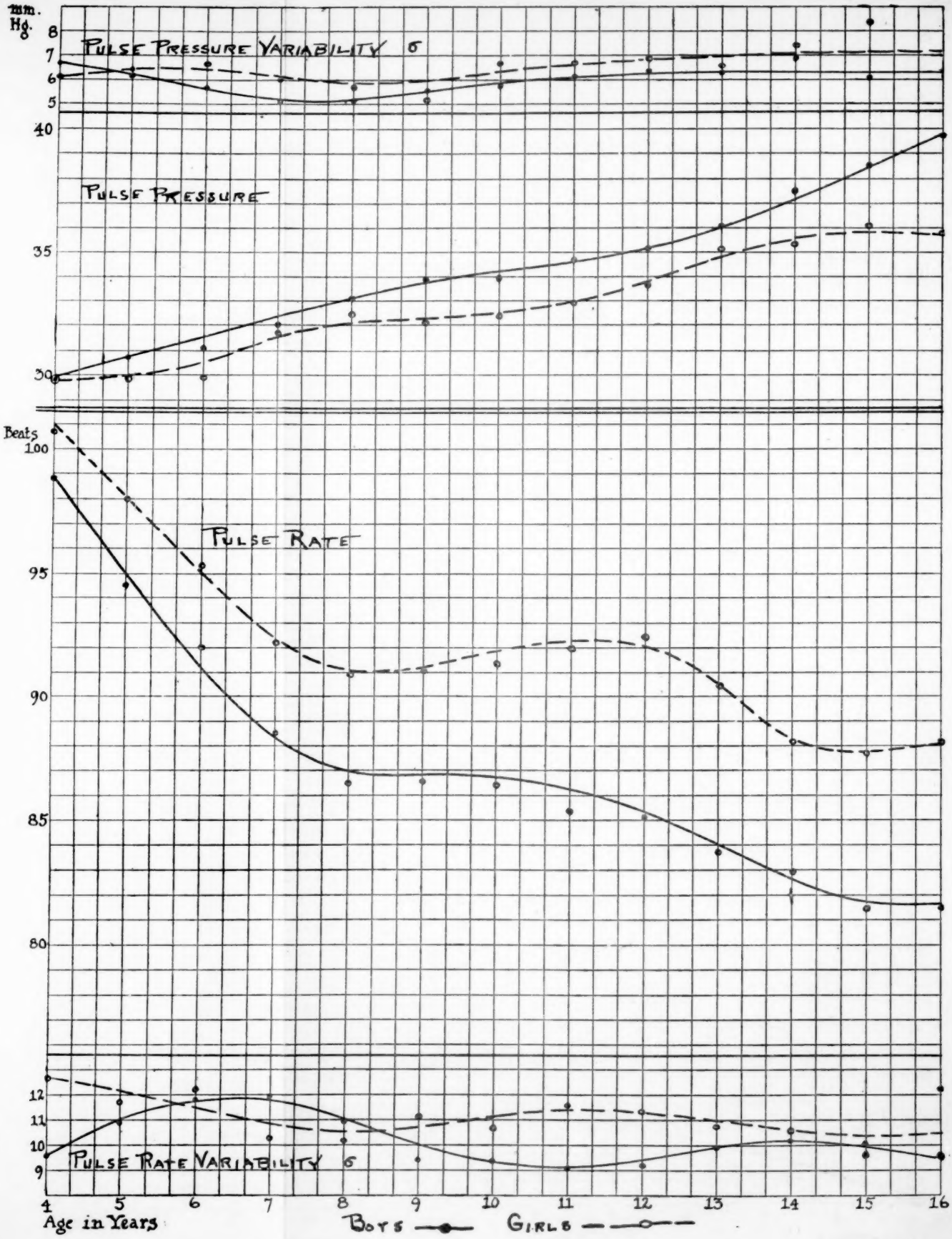

Fig. 5.—Pulse pressure and pulse, showing reciprocal relationship between the two.

TABLE 10.—Showing Rounded Values for Practical Use

Age, Years	Systolic Pressure			Diastolic Pressure				Pulse Pressure				Pulse Rate				Pulse Pressure × Pulse Rate			
	Boys Girls	Boys	Girls	Boys	Girls			Boys		Girls		Boys		Girls		Boys		Girls	
	Mean Mm.	σ Mm.	σ Mm.	Mean Mm.	σ Mm.	Mean Mm.	σ Mm.	Mean Mm.	σ Mm.	Mean Mm.	σ Mm.	Mean Beats	σ Beats	Mean Beats	σ Beats	Mean	σ	Mean	σ
4	89	5.5	6.5	60	7.0	60	6.5	30	6.5	30	6.0	99	10.0	101	12.5	2850	650	2900	700
5	92	6.0	6.5	62	7.5	62	6.5	31	6.5	30	6.5	95	10.5	98	12.0	2850	650	2900	700
6	94	6.5	7.0	64	7.5	64	7.0	31	6.0	30	6.0	92	11.5	96	11.5	2850	650	2900	650
7	97	6.5	7.0	65	7.5	66	7.5	32	6.0	32	5.5	89	11.5	96	11.0	2850	650	2900	650
8	100	6.5	7.0	67	7.0	68	7.0	33	6.0	32	5.5	86	10.5	92	10.5	2850	600	2900	650
9	101	6.5	7.0	68	6.5	69	7.0	34	5.5	32	5.5	87	10.0	91	10.5	2850	600	2900	650
10	103	6.5	7.0	69	6.0	70	6.5	34	5.5	32	6.5	86	9.0	92	11.0	2850	600	2900	750
11	104	6.5	7.0	70	5.5	71	6.5	35	6.0	33	6.5	85	9.0	93	11.0	2950	600	3000	800
12	106	6.5	7.0	71	5.0	72	7.0	35	6.5	34	6.5	85	9.5	93	11.0	2950	650	3100	800
13	108	6.5	6.5	72	5.0	73	7.5	36	6.0	35	7.0	84	10.0	91	11.0	3000	700	3200	800
14	110	6.5	6.5	73	5.0	74	8.5	38	6.0	35	7.5	83	10.0	89	10.5	3050	650	3150	800
15	112	7.0	7.0	75	5.5	76	9.5	39	6.0	36	7.5	81	10.0	88	11.0	3100	650	3100	750
16	115	7.0	7.5	76	6.0	79	10.0	40	6.0	35	7.5	81	9.5	90	11.5	3050	600	3000	700

To find deviation index: Find the difference between the actual reading and the average for age and sex; divide by σ (the standard deviation), keeping the sign throughout.

have adopted, and advise, in clinical application. Here we have used "rounded" values, giving our means in the nearest whole number to the "found" mean, and our standard deviations in the nearest half number (0.5), to the found value after another "smoothing." These assumed or rounded values are in every case within the limits of unreliability, within which the true value should fall. While they may be subject to later correction, they are, we believe, sufficiently accurate to justify their use in practice and to give significant results. The complete tabulations are shown in Tables 5, 6, 7, 8 and 9.

In each of these tables, the first column shows the mean (average) of the distribution; the second column, the limits of deviation from the "found" mean, within which the true mean probably occurs; the third column shows the standard deviation; and the fourth column, the limits of deviation from the "found" standard deviation within which the true standard deviation probably occurs.

Table 10 gives the rounded values for mean and standard deviation which we have tentatively adopted for actual use.

Figures 4 and 5 show graphically the changes that occur with age in average and variability of systolic pressure, diastolic pressure, pulse pressure and pulse.

COMMENT ON STANDARD CURVES

The application of the formulas for unreliability permits us to draw certain conclusions regarding the true or absolute values for our standards, which we offer tentatively.

EFFECT OF SEX

Systolic Pressure.—The mean systolic pressure for the two sexes varies only within the limits of unreliability, and, in the plotted curve the sexes run nearly the same course. It is evident that there is no significant difference in mean systolic pressure. The variation in standard deviation between the sexes is also within the limits of unreliability. Standard deviations, however, show very little age variation and we have tentatively assumed a sex difference because the mean (representing the most probable value of a series) standard deviation is about 6.5 mm. for boys and 7 mm. for girls. We feel safer in doing this because in all our other measurements—diastolic pressure, pulse pressure, pulse and pulse pressure—pulse product—the girls regularly show the same greater mean variability. That this phenomenon is the counterpart of a physiologically greater circulatory lability characteristic of the female sex seems to us highly plausible.

Diastolic Pressure.—A strict interpretation of the found values for diastolic pressure also shows the sexual differences to be within the limits of unreliability. We cannot, therefore, avoid the possibility that

there may be no actual sex difference. The form of the plotted curve, however, speaks strongly for the existence of an actual difference. After the fifth year the girls constantly show a higher mean pressure, which in some instances closely approaches the limits of unreliability. We have accordingly (subject to correction) assumed small differences after the sixth year in setting our standards. In the case of standard deviations, the situation is different. Here, after the eleventh year, the found differences are greater than the factor for unreliability, and we can definitely assert the presence of true differences. The variability of diastolic pressure in girls after the thirteenth year is very great and in marked contrast to the very small variability of the boys. That this variability in the girls is in some way connected with the vasomotor disturbances of adolescence seems to us to be beyond question.

Pulse Pressure.—Similar considerations may be applied to pulse pressure. Here the sex differences are within the limits of unreliability, but in the later ages so closely approach these limits and the differences throughout after the sixth year are so constantly in one direction, that it has been thought best to assume, tentatively, corresponding differences in the true values for both mean and standard deviation.

TABLE 11.—SHOWING ANNUAL RATE OF INCREMENT OF BLOOD PRESSURES, AND OF DECREMENT OF PULSE RATE

Age Group	Systolic		Diastolic		Pulse Pressure		Pulse Rate	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
4–9	2.3	2.5	1.5	1.9	0.8	0.5	2.5	1.9
9–13	1.7	1.7	1.0	0.9	0.6	0.8	0.7	0.1
13–16	2.6	2.0	1.2	2.0	1.2	—0.2	0.8	0.4

Pulse Rate.—Pulse rates after the sixth year show a divergence of means greater than the factor for unreliability, and we are justified in assuming the existence of true differences. The standard deviations do not differ by the amount of the factor, and may be identical. Here again, however, the difference is constantly in one direction.

Pulse Pressure × *Pulse.*—Pulse pressure times pulse is a measure, introduced by Erlanger and recently discussed by Wiggers, Addis and others, which may with several important reservations be regarded as a rough measure of cardiac output. Its main value is in estimating output in a given individual under different conditions of strain. In a large series the mean values may have some interest as indicating general tendencies. More than this we are unprepared to say, but we have calculated our data in the same way as for pressures and pulse. It is noteworthy that the pulse pressure-pulse product is practically constant up to the twelfth year and identical for the two sexes. At this time a slight progressive rise occurs which is distinctly greater for girls than

for boys. Whether this indicates a pre-adolescent or adolescent rise in cardiac output or merely reflects a lessening of peripheral resistance or some other circulatory change incidental to adolescence it is impossible to state with certainty.

TABLE 12.—Boys

Number	Age	Systolic, Mm.	Deviation Index	Diastolic, Mm.	Deviation Index	Pulse Pressure, Mm.	Deviation Index	Pulse	Deviation Index	Pulse Pressure × Pulse Rate	Deviation Index	Remarks
1	3	102	+2.4	52	+1.1	50	+3.1	106	+0.7	5300	+3.8	Bronchial asthma, eczema, malnutrition
2	5	88	−0.8	66	+0.5	22	−1.4	114	+1.8	2508	−0.5	Convalescent from pneumonia
3	6	100	+0.9	78	+1.9	22	−1.5	120	+2.4	2640	−0.3	Convalescent from pneumonia
4	6	110	+2.5	64	0.0	46	+2.5	138	+4.0	6348	+5.4	Convalescent from measles and whooping cough
5	6	76	−2.8	56	+1.1	20	−1.8	...	.....	....	.....	1/27/20; bronchial asthma
		92	−0.3	62	−0.3	30	−0.2	100	+0.7	3000	+0.2	3/23/20; same pateint after 7 weeks in country without attacks
6	7	82	−2.3	66	+0.1	16	−2.7	...	.....	....	.....	Eczema, chronic bronchitis (asthma?)
7	8	82	−2.8	72	+0.7	10	−3.8	108	+2.1	1080	−3.0	Imbecile; cold, blue hands
8	9	86	−2.3	60	−1.2	26	−1.4	106	+1.9	2756	−0.2	3/16/20; bronchial asthma
		86	−2.3	54	−2.2	32	−0.4	108	+2.1	3456	+1.0	3/23/20; same patient
9	9	80	−3.2	70	+0.3	10	−4.4	...	.....	....	.....	1/27/20; bronchial asthma, cyanosis
		92	−1.4	60	−1.2	32	−0.4	100	+1.3	3200	+0.6	2/ 3/20; same patient
		98	−0.5	84	+2.5	14	−3.6	96	+0.9	1344	−2.5	2/24/20; same patient
		96	−0.8	64	−0.6	30	−0.7	90	+0.3	2700	−0.3	3/ 2/20; same patient
10	9	102	+0.1	80	+1.8	22	−2.2	80	−0.7	1760	−1.8	2/17/20; bronchial asthma, no attack for 3 weeks
		96	−0.8	64	−0.6	32	−0.4	96	+0.9	3072	+0.4	3/9/20; same patient; no attack for 6 weeks
11	9	68	−5.1	54	−2.2	14	−3.6	...	.....	....	.....	Frequent headaches, pale, cold hands
12	10	114	+1.7	46	−3.8	68	+6.2	80	+0.7	5440	+4.4	Severe postural defects; very poor muscular tone; malnutrition
13	11	94	−1.5	66	−0.7	28	−1.1	82	−0.3	2296	−1.1	Bronchial asthma
14	12	120	+2.2	74	+0.6	46	+1.4	116	+3.3	5346	+3.7	"Nervous," pale, mother luetic
15	13	90	−2.8	50	−4.4	40	+0.7	...	.....	....	.....	Froelich syndrome, obese
16	13	80	−4.3	48	−4.8	32	−0.7	72	−1.2	2304	−1.0	2/3/20; bronchial asthema
		88	−4.3	54	−3.6	88	−1.3	72	−1.2	6336	+4.8	3/2/20; same case
17	13	88	−3.1	80	+1.6	8	−4.7	104	+2.0	832	−3.1	Congenital heart disease, probably pulmonic stenosis; decompensation with extreme cyanosis; died about 1 week later
18	14	140	+4.0	58	−3.1	82	+7.1	92	+1.1	7544	+6.2	Vasomotor instability; blue, cold hands; heart normal except for powerful apex thrust and booming first sound; urine repeatedly normal; has collapsed several times after exercise

EFFECTS OF ADOLESCENCE: PERIODIC VARIATIONS

Our graphs show throughout certain fairly constant periodic variations which we are inclined to believe correspond with periods in the development of children. Similar fluctuations occur, it will be recalled, in curves of mean height and weight. Expressed in terms of annual increment, the fluctuations are shown in Table 11.

Both diastolic and pulse pressures, beginning at about age 9, show a period of standstill which lasts until about the twelfth year, when the rate of increase is again accelerated. During the corresponding periods the curve for pulse is in the reverse direction, the rate of decrease in

TABLE 12.—GIRLS

Number	Age	Systolic, Mm.	Deviation Index	Diastolic, Mm.	Deviation Index	Pulse Pressure, Mm.	Deviation Index	Pulse	Deviation Index	Pulse Pressure × Pulse Rate	Deviation Index	Remarks
19	7	62	−5.0	48	−2.4	14	−3.3	84	−1.1	1176	−2.7	Enuresis; "nervous"; speech defect
20	10	82	−3.0	70	+0.1	12	−3.1	106	+1.3	1272	−2.2	Eczematous blepharitis; blue hands; poor muscular tone
21	11	82	−3.1	66	−0.8	16	−2.6	88	−0.5	1408	−2.0	Vasomotor instability; red-purplish hands; styes
22	11	112	+1.1	48	−3.5	64	+4.8	120	+2.5	7680	+5.9	Vasomotor instability; mottled hands; well nour.
23	12	130	+2.0	70	−0.3	50	+2.5	90	−0.3	4500	+1.8	Frequent colds; tuberculosis (?); anemia
24	12	92	−2.0	60	−1.7	32	−0.3	104	+1.0	3328	+0.3	Bronchial asthma; chronic mediastinitis; malnutrition
25	12	72	−4.9	60	−1.7	12	−3.4	102	+0.8	1224	−2.4	Chronic general lymphadenitis; poor vasomotor tone
26	13	128	+3.1	86	+1.7	42	+1.0	92	−0.1	3864	+1.0	Chronic endocarditis; chorea
27	13	102	−0.9	56	−2.3	46	+1.6	124	+3.0	5704	+3.1	2/17/20; chronic endocarditis, mitral insufficiency
		100	−0.8	56	−2.3	44	+1.3	120	+2.6	5280	+2.6	3/11/20; same patient
		96	−1.8	56	−2.3	40	+0.7	100	+0.8	4000	+1.0	5/29/20; same patient
28	14	114	+0.6	54	−2.4	60	+3.3	76	−1.2	4560	+1.8	Frequent headaches; palpitation; poor vasomotor tone
29	14	110	0	52	−2.6	58	+3.1	78	−1.0	4524	+1.7	Vasomator instability; chilblains
30	15	94	−2.6	66	−1.0	28	−1.1	108	+1.8	3024	−0.1	1/31/20; mitral insufficiency, well compensated; poor vasomotor tone; blue hands; migraine; periodic vomiting
		98	−2.0	66	−1.0	32	−0.5	100	+1.0	3200	+0.1	2/7/20; same patient
31	16	118	+0.4	96	+1.7	22	−1.7	124	+3.0	2728	−0.4	2/10/20; chronic endocarditis, mitral stenosis and insufficiency; malnutration
		106	−1.2	72	−0.7	34	−0.1	106	+1.4	3604	+0.9	3/23/20; same patient
32	15	88	−3.4	62	−1.5	16	−2.7	82	−0.5	1272	−2.5	Dysmenorrhea; constipation; "always tired"

pulse rate coming to a halt in the case of boys and in the case of girls being replaced by a period of increase; in both, this period is followed by a period of decrease. The changes in the diastolic pressure and pulse pressure are, therefore, balanced by corresponding changes in the pulse rate in the opposite direction and this balance is clearly shown by the relative constancy of the pulse pressure-pulse product.

Such fluctuations are evidence of changes in the circulation incident to bodily development and illustrate the periodic character of development. It is probable that they indicate functional and, perhaps, organic changes in the vessels and in their nervous control, causing various inner readjustments in circulation. In any case it appears that preceding adolescence there is a period of relative inactivity, both in growth and

in the rapidity of changes in circulatory phenomena, which is followed by a period of acceleration in these various reactions.

STATES OF PATHOLOGIC TENSION

While we have in the main confined ourselves up to the present time, to the study of normal children, we have made a few scattered observations in children suffering with various disorders and the results, shown in Table 12, will serve to illustrate the application of the methods under discussion.

Attention is directed to the rather interesting findings in children with asthma and the fact that in some cases improvement in condition can be followed by a lessening of the deviation index.

It will be recalled that the statement is often made, and can be found in at least one of the important text-books on pediatrics, that the study of blood pressure in children is of little practical importance. So long as sphygmomanometry is only used qualitatively to determine the presence or absence of permanent hypertension, such an assertion may be true in general. Even so, we have seen a few cases in which abnormally high systolic pressure gave us the first "lead" to a diagnosis. In the field of hypotension in children there appears to be much unexplored ground. The vasomotor disorders are among the most significant incidents of childhood, particularly during adolescence in girls and by no other method is their presence so clearly revealed as by diastolic and pulse pressures. In asthma and the allied disorders blood pressure changes certainly have a significance not as yet fully appreciated. In the nephritis of children Gordon,[14] and Berkley and Lee,[15] have noted significant alterations in pressure. In severe diarrheas Trumpp[16] has noted a marked drop in systolic pressure. Rolleston[17] has noted very subnormal systolic pressures in diphtheria reaching the low point during the second week and gradually returning to normal by the seventh week. He also noted that during convalesence the pressure in the recumbent position was higher than in the erect position. That further observations of blood pressure would give us information of value as to the progress of myocardial degeneration in diphtheria seems to us almost certain; yet Rolleston concludes his paper by saying that the method is of little practical value. If further evidence of the

14. Gordon, L.: The Blood Pressure in Cases of Acute Nephritis of Children, Arch. Pediat. **28**:343, 1911.

15. Berkley, H. K., and Lee, J. M.: Hypertension in Nephritis in Childhood, With a Study of Ninety-Three Cases, Am. J. Dis. Child. **13**:354 (April) 1917.

16. Trumpp, J.: Blutdruckmessungen an gesunden und kranken Säuglingen, Jahrb. f. Kinderh. **63**:43, 1906.

17. Rolleston, J. D.: The Blood Pressure in Diphtheria, Brit. J. Dis. Child. **8**:433, 1911.

value of blood pressure determinations in children were needed, we might point to the recent paper of Wilson [18] who used systolic pressures as an index of cardiac fatigue from strain.

Finally, mention should be made of the recent contribution by Alvarez and his associates [19] in which the systolic pressures of a large group of adults were studied by the frequency method. A comparison of their results with ours is of much interest.

The general usefulness of the method we have discussed may seem to be limited by the apparent difficulties of application. The size and apparent complexity of the tables can hardly be avoided. It is necessary to use Table 11 at least, in applying the method, and we see no other way at present further to simplify it. The effect of age and sex both on averages and on deviation values in childhood is so great that the exact standard figures must be at hand before the index can be determined. As contrasted with the difficulties the advantage of being able to assign to a given reading the place which it occupies in the normal scale and of being able to state in objective terms the degree of normality or of abnormality seems worth some extra effort, especially to the pediatrist who has vainly endeavored to estimate the true significance of his blood pressure findings and very likely has abandoned the measurement of blood pressure in children as of no practical value. We hope we have not made the method look too formidable. In reality its application is very simple. To find the deviation index requires only about 20 seconds, and its significance can be determined by a glance at Table 1, or can be remembered after a little practice. Because the deviation index itself lacks immediate significance on inspection until one has worked with it for some time, it might be better instead to record the corresponding "chances" or "betting odds," with their striking imaginative appeal, though this would add another step in the process.

We do not wish to set up our standards as in any way definitive or final. We have attempted to express the probable accuracy or reliability in Tables 6-10. When a larger series of normal data has been gathered, a closer approximation to the true or absolute values will be possible. Till then we believe that our standards will suffice for practical purposes to define and analyze the significance of blood pressure findings in chidren, in the hospital, clinic and office, both in individual cases and in the study of series of cases showing functional or anatom-

18. Wilson, M. G.: The Circulatory Reactions to Graduated Exercise in Normal Children, Am. J. Dis. Child. **20**:188, 1920.

19. Alvarez, W. C., Wulzen, R., Taylor, F. B. and Starkweather, E.: Blood Pressure in University Freshmen and Office Patients, Arch. Int. Med. **26**: 381, 1920.

ical aberration of the cardiovascular apparatus. By using the method, too, it is possible that more reliable tests of cardiovascular function may be developed.

SUMMARY

1. Our lack of a proper measure of the normal range of blood pressure and of the relative significance of various deviations from the average normal blood pressure is due to the failure to apply correct methods to the compilation of normal statistics and has made it impossible in practice to obtain the full usefulness inherent in sphygmomanometric methods. Our need is for standards of deviation.

2. Such standards must be based on the statistical method of frequency distribution. In connection with normal averages they can be used in the individual case to determine the relative significance and importance of any deviation from the normal.

3. Normal means and standard deviations for systolic, diastolic and pulse pressures and for pulse and pulse pressure—pulse product have been compiled for boys and girls separately, by year, between the ages of 4 and 16 years.

4. Reference tables for clinical application are given.

5. Mean systolic pressure shows no significant difference between the sexes within the period studied. Mean diastolic and mean pulse pressure do show significant sexual differences. Standard deviations are in practically all cases greater for girls, indicating a normally greater variability in female children, which is particularly marked during adolescence.

6. Illustrative examples of the method of measuring deviations in various pathologic states are given.

7. The frequent occurrence of states of hypotension in children is pointed out, in contrast with the relatively greater frequency of states of hypertension in adults. Note is made of the common occurrence of hypotension in bronchial asthma in children.

8. The method, while involving the use of the reference tables, requires only a few seconds to apply.

THE TONSILS AND SCARLET FEVER*

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The observation of many patients suffering with scarlet fever gave me the impression that their fate depended, in part on the anatomic configuration of their throats, and led me to verify this conclusion by statistical study. To this end special charts were printed, and observations were collected, covering certain definite points in tonsil structure. I am indebted to assistant resident physicians and interns in the scarlet fever pavilion of the Willard Parker Hospital, especially to Doctors Cootner, Tolle and Crawford, who made the observations on admission. A large number of the observations were verified or corrected by me on my later visit. It is thought that in this way the observations have greater value as averaging any personal error.

In table 1 are tabulated 154 cases which were observed during my services in 1919 and 1920. This does not represent all the cases seen during that time, as cases in which inadequate observations were made have been discarded. The chart has been prepared showing the sex, age, number of days ill and the condition on admission. "Very sick" describes the condition of any patient with an acute infection who is not so desperately ill that the prognosis is doubtful. Under rash, the number of crosses indicate in a rough way the intensity of the rash. A mild punctate erythematous rash, sufficient to establish a diagnosis of scarlet fever, is indicated by a single cross; two crosses indicate that the rash was well developed; three crosses that the rash was quite intense and four crosses that rash is so intense that the description "boiled lobster" is applicable. By the size of the tonsil is indicated our approximate judgment based in the case of covered tonsils on the amount of bulging into the anterior pillar. By small tonsils we designate a tonsil which is not larger than a filbert or hazel nut; the large tonsils appear to be the size of a walnut or larger. Moderate sized tonsils are intermediate. Tonsils are described as buried when they are covered by the anterior pillar, by a fold of mucous membrane, by a fold of plica or by the posterior pillar. Such tonsils may give the appearance of being concealed behind an iris diaphragm. The major portion of their surfaces is unexposed.

Adhesion to the pillars indicated that on swallowing, the tonsils do not lie free in the fossa but are apparently flush with the pillar and

* Received for publication, Dec. 26, 1920.

TABLE 1.—ANALYSIS OF CASES

Number	Hospital Number	Sex	Age, Yrs.	Days Ill	Condition on Admission	Rash	Tonsil Size	Adhesion to Pillar	Compressed on Swallowing	Lymph Nodes		Difference	Character of Case	Complications and Remarks	Result
										Anterior	Posterior				
1	2096	♀	12	3	V.S. bronchitis	+++	L	—	—	—	—	—	Mild	Temp. to 101 F. in 4th week....	R
2	2103	♀	15	3	V.S.	++	L	+	—	+	+	—	Mild	Slight adenitis	R
3	2101	♀	4	3	V.S.	+	L	+	+	++	+	—	Moderate	Adenitis moderate; Temp. 100 F. until 3d week	R
4	2102	♂	2	2	V.S.	++	L	—	..	..	..	—	Moderate	.....	R
5	2111	♀	7	2	V.S.	+	S	—	—	++	+	—	Moderate	Adenitis; Temp. to 101.8 F. in 2d and 4th weeks	R
6	2136	♂	29	2	V.S.	++	L	+	—	++	+	—	Moderate	Hemic murmur	R
7	2140	♀	7	1	V.S.	++	L	+	±	++	+	—	Moderate	.....	R
8	2152	♀	9	2	P.D.	+++	..	—	..	++	+	—	Moderate	.....	R
9	2172	♂	8	3	V.S.	++	L crypts	—	..	++	+	—	Moderate	.....	R
10	2205	♂	4	?	V.S.	++	V L	..	+	—	—	—	Severe	Ethmoiditis; sinus thrombosis; convulsions; Temp. 101.4 F. four days before nodes enlarged	†
11	2231	♀	3	4	V.S.	++	L	+	+	+++	+	—	Moderate	Right cervical adenitis.....	R
12	2232	♀	7	6	V.S.	+++	S	—	— free in fossa	+	+	—	Moderate	.....	R
13	2281	♀	20	5	V.S.	+	S	—	— free in fossa	+	+	—	Mild	.....	R
14	2288	♂	6	1	V.S.	++	L	+ tightly buried encapsulated	+	+	+	—	Severe	Cardiac arrhythmia; foot abscess	R
15	2300	♂	6	4	V.S.	+	L	+ encapsulated, buried	+	+	+	—	Moderate	Abrasion on forehead, fell from bed	R
16	2308	♂	9	'	V.S.	+	S	+	..	++	+	—	Mild	.....	R
17	2309	♂	5	4	V.S.	+	S	—	..	++	+	—	Mild	Infected vaccination in 2d week	R
18	1961	♂	2	2	V.S.	+	L	—	+	+++	+	—	Mild	Cervical adenitis	R
19	1979	♀	4	2	V.S.	++	L	—	+	++	+	—	Mild	Cervical adenitis; Temp. 103 F. 2d week	R
20	1981	♀	3	2	V.S.	++	L	+	++	+++	+	—	Severe	Cervical adenitis; right facial paralysis; bronchopneumonia; diphtheria	†
21	1990	♀	8	3	V.S.	++	L	—	..	++	+	—	Mild	.....	R
22	2031	♀	7	4	V.S.	++	L	+ tonsillotomy	..	+	+	L>R	Mild	Chickenpox	R
23	2035	♂	7	?	V.S.	+	L	+ tonsillotomy	..	++	+	—	No case ?	Pneumonia and diphtheria.....	R
24	2037	♂	4	5	V.S.	+	L	—	..	+	+	..	Severe	Diphtheria; discharged against advice	R
25	2052	♀	7	2	V.S.	++	L	—	—	++	+	L covered with exudate	Mild	Adenitis; left peritonsillar abscess, drained	R
26	2053	♂	16	3	V.S.	+	S	+ tonsillectomy tabs	..	..	..	..	V mild	.....	R
27	2264	♀	14	9	V.S.	—	S	+	+	++	+	—	V mild	.....	R
28	2651	♂	9	3	V.S.	+	L	+ buried	+	+	+	—	Mild	.....	R
29	2664	♂	7	2	V.S.	++	L	—	--everted	+	+	—	Mild	Whooping cough	R

30	2701	♂	6	[illegible]	V.S.	+	L	+ buried	—	++	+	—	Mild	.....	R
31	2716	♀	3	2	V.S.	+	L	+ buried	+	++	+	—	Moderate	Double otitis media purulenta	R
32	2726	♀	6	4	V.S.	++	L	+ buried	+	++	++	L>R	Moderate	Lymphadenitis; Temp. on 10th day for 3 days	R
33	2727	♀	8	1	V.S.	+	S	+	+	++	++	—	Moderate	.....	R
34	2733	♂	25	3	V.S.	+++	S	+ buried	+	++	..	..	Moderate	Considerable cervical adenitis..	R
35	2737	♀	28	4	V.S.	++	L cryptic	+ buried	+	+	+	L>R	Moderate	Double cervical adenitis; general arthritis relieved by salicylate	R
36	2738	♂	14	3	V.S.	++	L	+ buried	+	++	++	—	Moderate	Double cervical adenitis; left axillary adenitis; Temp. 100 F. 12th to 16th day	R
37	2739	♂	9	3	V.S.	+	S tonsillectomy, remnant on rt side	0	..	++R	+	L remnant	Mild	.....	R
38	2759	♀	8	1	V.S.	++	0	0	..	+	+	..	Mild	.....	R
39	2760	♀	28	3	V.S.	++	S	+ buried	—	+	+	—	Moderate	Slight temperature persisted 16 days	R
40	2765	♀	22	3	V.S.	++	S tonsillotomy 10 years previously	+ buried	+	++	++	—	Moderate	R. follicular tonsillitis on 12th day; temperature rise	
41	2782	♀	18	3	V.S.	++	S	+ buried	+	++	++	—	Moderate	Left cervical adenitis; Temp. to 101 F. for 16 days	R
42	2783	♂	8	2	V.S.	++	L	+ buried	..	+	+	—	Moderate	Bilateral otitis media purulenta	R
43	2791	♀	23	2	V.S.	++	Moderate	— buried	+	+R	+	R slightly compressed	Moderate	.....	R
44	2796	♂	7	4	V.S.	—	L cryptic	+ buried	+	+	+	L>R	Moderate	Cervical adenitis; follicular exudate 27th day	R
45	2806	♀	21	1	V.S.	++	S remnant tonsillectomy	—	—	+	+	—	Mild	.....	R
46	2817	♂	9	4	P.D.	+++	L	+ buried covered with plica	—	+	+	—	Moderate	Rise of temperature on 14th day to 102 F.	R
47	2833	♀	4	4	V.S.	—	L	+ buried	+	+	+	—	Mild	.....	R
48	2867	♂	9	2	V.S.	++	L +	+ buried covered with plica	+	++	++	—	Moderate	Temp. normal 6th day; severe cervical adenitis; received antitoxin because of clinical appearance	R
49	2531	♂	27	6	V.S.	+	L	L buried R free	—	..	..	L buried R free	Mild	Lateral labial herpes	R
50	2541	♂	2	1	V.S.	++	L	+ buried	+	+	+	—	Severe	Temp. 101 F. for week, reached 104 F. in 2 weeks; pneumonia and death 19th day	†
51	2586	♂	5	2	V.S.	++	M	+ buried covered with plica	+	++	++	—	Severe	Temperature high; bronchopneumonia 2d week	†
52	2480	♂	5	3	V.S.	+	L	+ cryptic	..	+	+	Exudate on L tonsil	Moderate	Nasal discharge; chickenpox....	R
53	2462	♀	12	2	V.S.	++	L	L bulging beyond pillar	—	+	++	L>R	Mild	.....	R

V.S. = very sick; P.D. = prognosis doubtful; C. = convalescent; crosses in "Rash" column indicate intensity of rash.

TABLE 1.—Analysis of Cases—(*Continued*)

Number	Hospital Number	Sex	Age, Yrs.	Days Ill	Condition on Admission	Rash	Tonsil Size	Adhesion to Pillar	Compressed on Swallowing	Lymph Nodes		Difference	Character of Case	Complications and Remarks	Result
										Anterior	Posterior				
54	2493	♂	7	2	V.S.	+	L	+	+	..	++	—	Moderate	Cervical adenitis; Temp. 104 F. 6th day	R
55	2513	♀	9½	2	V.S.	+	M	R + buried L —	..	+	..	R adherent L free	Moderate	.....	R
56	2523	♂	24	1	V.S.	++	M	R — L + buried	+	++	++	R free L buried	Moderate	Right cervical adenitis; bradycardia	R
57	2524	♂	5	1	V.S.	++	L	+ buried	..	++	++	—	Moderate	Adenitis; temperature rose to 101 F. occasionally during convalescence	R
58	2529	♂	9	2	V.S.	++	S previous tonsillectomy	— rem. on R none on L	..	Only on R	Only on R	On R	Moderate	Temp. 103 F. on 5th day; adenitis	R
59	2559	♀	6	2	V.S.	+++	L	+ not buried	—	++	++	..	Severe	Adenoidectomy; tonsillectomy; nephritis; meningitis	†
60	2572	♀	11	2	V.S.	++	L	Buried	—	—	++	..	Moderate	.....	R
61	2584	♀	11	3	V.S.	++	L	L + R —	..	L + R—	—	L + R—	Moderate	.....	R
62	2587	♀	4½	3	V.S.	++	L	+ buried	+	++	..	L>R	Moderate	Double cervical adenitis; L>R	R
63	2605	♀	12	2	V.S.	++	VS	Almost completely buried	..	+	++	—	Moderate	Slight R. cervical adenitis.....	R
64	2612	♀	22	3	V.S.	++	Tonsillectomy Mod.	..	..	+	..	—	Moderate	Rheumatism (also "e flu"; teeth abscessed and capped	R
65	2613	♀	2	2	V.S.	++		+ buried	+	R++ L+	..	—	Moderate	Developed diphtheria, 6.000 c.c. antitoxin	R
66	2520	♂	5	2	V.S.	++	Previous tonsillectomy	—	..	L small	..	—	Mild	.....	R
67	2525	♂	4	1	V.S.	++	L	+ buried	+	++	++	—	Moderate	Nasal discharge; moderate double cervical adenitis; bilateral OMP; chickenpox	R
68	1971	♀	9	2	V.S.	++	S	— buried	—	+	+	—	Mild	Herpes (lateral)	R
69	1995	♀	5½	5	V.S.	+	S plica	— buried	—	+	+	—	Mild	.....	R
70	1999	♀	12	3	V.S.	++	L plica	+	+	+	+	—	V mild	.....	R
71	2002	♂	4	2	V.S.	++	L plica	+	..	+	+	L worse	Moderate	Cervical adenitis on admission; left adenitis, diphtheria, Temp. 102 F. 3d week	R
72	2003	♀	4	4	V.S.	+	M plica	—	—	—	—	—	Moderate	No adenitis; foot infection caused rise of temperature	R
73	2023	♀	10	4	V.S.	++	S plica	—	—	++	—	—	Moderate	Arrhythmia 3d week; acute tonsillitis, double cervical adenitis 3d and 4th weeks	R
74	2040	♀	7	7	V.S.	..	L	—	—	+	+	—	Mild	Vacillating temperature 2d week	R
75	2052	♀	17	3	V.S.	++	L	—	—	+	—	—	Mild	No adenitis	R
76	2091	♀	3½	1	V.S.	++	L	—	—	+	+	R>L	Mild	.....	R
77	2100	♀	9	10	V.S.	..	R large L small	R + L — covered with plica	—	+	+	R>L	Mild	.....	R
78	2129	♀	9	2	V.S.	++	S	— buried and covered with plica	—	+	—	—	Severe	Wrist arthritis with temperature; 2d and 3d weeks cervical adenitis	R

79	2138	♀	22	2	V.S.	++	S	— plica	—	—	—	—	Mild	.....	R
80	2152	♂	4	2	V.S.	++	L	+ plica	—	+	+	—	Moderate	Empyema on admission; adenitis	R
81	2165	♀	30	2	V.S.	++	L plica	+	—	+	+	L>R	Mild	Peritonsillar abscess with adenitis; tonsillectomy 2d week	R
82	2179	♀	7	2	V.S.	++	L plica	+	+	+	+	R larger	Mild	.....	R
83	2194	♂	22	6	V.S.	++	S plica	+	—	+	+	—	Mild	.....	R
84	2250	♂	13	2	V.S.	+++	L plica	+	+	+	+	—	Severe	.....	R
85	2212	♂	6	3	V.S.	++	L plica	— buried	+	++	+	—	Severe	Marked double cervical adenitis; double OMP and mastoiditis; P. D. 5th day	†
86	2234	♀	21	2	V.S.	++	M partly covered	—	—	—	—	—	Mild	.....	R
87	2255	♀	?	3	V.S.	++	S almost covered	—	—	—	—	—	Mild	.....	R
88	2273	♀	5	5	V.S.	++	S buried	+	+	+	+	R>L	Severe	Adenitis; profuse nasal discharge; tonsillectomy refused 8th day; died 15th day	†
89	2327	♀	2	3	V.S.	++	L plica	+	—	++	++	—	Severe	Cervical adenitis; nasal discharge; ulcer of mouth	†
90	2544	♂	20 m.	2	P.D.	++	L	+	+	++	++	—	Severe	Profuse nasal discharge 3d day	†
91	2244	♀	6	2	V.S.	++	M almost covered	+	..	+	+	—	Moderate	.....	R
92	2268	♀	24	2	V.S.	++	M	+ R tonsil with plica	..	..	..	..	V mild	.....	R
93	2271	♂	8	2	V.S.	++	Tonsillectomy	..	..	SI +	+	—	Mild	Temperature due to infected hernia	R
94	2272	♀	14	2	V S.	++	L plica	+	..	+	+	—	Moderate	Temperature persisted 1 week..	R
95	2279	♂	29	2	V.S.	++	M cryptic	+ covered with plica	..	few	..	—	Mild	Bradycardia	R
96	2294	♀	3½	2	V.S.	++	L	L + R — buried	..	+	+	L buried R free	Mild	Developed diphtheria	R
97	2312	♂	22	3	V.S.	++	L plica	+	..	+	+	R>L	Moderate	Peritonsillar abscess 20th day; tonsillectomy under local anesthesia 21st day	R
98	2341	♂	6½	3	V.S.	+	S	+	—	++	++	—	Moderate	.....	R
99	2360	♀	4½	1	V.S.	+	S	—	..	+	+	—	Mild	.....	R
100	2383	♀	24	5	P.D.	++	M	—	..	+	+	—	V mild	Lateral herpes	R
101	2385	♀	21	2	V.S.	++	L plica	+	..	+	..	R>L	Mild	.....	R
102	2438	♀	10	8	V.S.	+	M plica	..	..	+	+	—	V mild	.....	R
103	2263	♀	5	4	V.S.	++	M plica	Buried	..	+	+	—	Mild	Diphtheria	R
104	2293	♂	3	1	V.S. then P.D.	++	L plica	— large crypts	..	+	+	—	Severe	Bilateral cervical adenitis and bilateral otitis media; died 6th day	†
105	2326	♀	6	1	V.S.	++	Tonsillectomy	..	..	+	+	..	Moderate	.....	R
106	2361	♂	2	1	V.S.	++	L	+	+	++	++	—	Severe	Bilateral otitis media; adenitis; profuse nasal discharge; plica incised; frequent Temp. 104 F.	R
107	2374	♂	3	2	V.S.	++	L buried	+ covered	..	+	+	—	Mild on adm. sev.	Bronchopneumonia. cervical adenitis; death 15th day	†
108	2431	♂	5	4	V.S.	++	Tonsillectomy	..	..	..	..	—	Mild	Herpes (lateral)	R

TABLE 1.—Analysis of Cases—(*Continued*)

Number	Hospital Number	Sex	Age, Yrs.	Days Ill	Condition on Admission	Rash	Tonsil Size	Adhesion to Pillar	Compressed on Swallowing	Lymph Nodes		Difference	Character of Case	Complications and Remarks	Result
										Anterior	Posterior				
109	2435	♂	8	1	V.S.	++	Tonsillectomy	..	..	..	++ Rt. only	R < L	Mild	Nasal discharge	R
110	2439	♂	6	7	V.S.	+	L	+ to ant. pillar	..	+	..	—	Mild	.....	R
111	3334	♂	12	2	V.S.	++	L ½ buried	..	+	..	..	..	Mild	Bilaterally exudate, Schick intravenous +, 25,000 units	R
112	3329	♂	20	3	V.S.	++	L buried	— covered	..	.	..	..	Moderate	.....	R
113	3304	♀	7	2	V.S.	++	VL buried	— covered	..	..	..	..	Moderate	Temp. 99-100 F. for weeks; intravenously 25,000 units	R
114	3269	♀	8	2	V.S.	++	ML strep. exudate	..	—	..	..	..	Moderate	Temperature normal 6th day.	R
115	2875	♂	7	3	?	++	L cryptic	—	—	..	+	..	Moderate	.....	R
116	2902	♀	25	3	V.S.	++	L buried	..	+	+	..	..	Moderate	General joint pains 7th day; bradycardia 3 days; salicylates; relief	R
117	2904	♀	4	2	V.S.	++	M buried	+	+	..	+	..	Moderate	.....	R
118	2934	♂	4½	1	V.S.	..	Previous tonsillectomy	..	..	..	..	..	Moderate	Operated for inguinal adenitis 8 days before admission	R
120	3441	♀	3	1	V.S.	++	L buried	..	..	..	..	..	Moderate	Cervical adenitis L>R; arrhythmia; temperature 12 day	R
121	3434	♂	6	1	V.S.	++	L	..	..	..	..	..	Moderate	Bradycardia lasting untill 16th day; nasal discharge; myocarditis	R
122	2571	♂	13	2	V.S.	++++	L buried	+	+	++	..	—	Severe	Diphtheria, antitoxin 2d day; albuminuria; bilateral OMP	R
123	2604	♂	5	2	V.S.	++	M buried	..	—	+	..	—	Moderate	Infected plastic on hand; large glands drained before admission; chicken pox; nasal discharge; bilateral OMP	R
124	2065	♂	7	3	V.S. incised glands on neck on adm.	++	Previous tonsillectomy	..	..	—	—	..	Mild	.....	R
125	2099	♂	2	2	V.S.	++	L buried	—	+	+	+	..	Severe	Developed measles and chicken pox	R
126	2105	♀	5	4	V.S.	++	L buried by plica covered	+	..	++	..	..	Severe	Double cervical adenitis; required incision	R
127	2178	♂	5	17	V.S.	At home 17 days	L buried	+	+	+	+	—	Severe	Nephritis	R
128	2211	♀	6½	1	V.S.	++	L plica	..	..	++	..	—	Severe	Double OMP; mastoiditis operation	R
129	2243	♂	16	3	V.S.	++	L cryptic	—	..	+	+	—	Moderate	Slight cervical adenitis.....	R
130	2307	♂	4½	2	V.S.	+	S buried	— +	..	..	+	R adherent	Moderate	No adenitis; temperature 14 days; developed measles and bronchitis	R
131	2325	♂	3½	3	V.S.	++	M cryptic plica	+	+	++	+++	—	Severe	Measles; cervical adenitis.....	R

132	2346	♂	8	7	V.S.	++	VL	R + L −	+	++	..	..	Moderate	OMP; developed varicella.....	R
133	2364	♀	6	5	V.S. bron.pn.	++	S buried plica	..	+	++	++	—	Severe	OMP; measles	R
134	2384	♀	14	3	V S.	++	S buried	—	..	+	L>R	—	Moderate	OMP; general bronchitis.....	R
135	2628	♂	12	1	V.S.	+	Tonsillectomy	..	..	+	—	..	Mild	.....	R
136	2637	♀	13	4	V S.	+	Tonsillectomy	..	..	—	—	..	Mild	.....	R
137	2644	♀	10	2	V.S. empyema	++	L fibrous	+	—	+	++	..	Moderate	Had empyema on admission...	R
138	2645	♀	30	2	V.S.	++	S buried	..	+	1 gland on left	..	L>R	Moderate	Temp. 12 day; arthritis relieved by acetylsalycilic acid	R
139	3368	♀	7	2	V.S.	++	L irreg.	..	..	..	..	..	Moderate	.....	R
140	2317	♂	7	2	V.S.	++++	Tonsillectomy	..	..	—	—	..	Moderate	Temp. normal 5th day.....	R
141	3000	♂	12	?	P.D.	+++	Follicular exudate bilateral	+	+	+	..	..	Moderate	Peritonsillar infiltration; Temp. normal 6th day	R
142	3016	♂	6	1	V.S.	++	L Hyper. on both	..	—	..	..	..	Mild	Temp. normal 3d day.....	R
143	3009	♂	2½	3	V.S.	++	S embedded, mostly removed	..	..	+	+	More on left	Moderate	Temp. normal 14th day.....	R
144	3101	♂	3½	3	P.D.	++	L buried hypertr.	..	+	+	+	..	Severe	Left cervical adenitis 21st day; Temp. up till 24th day; nasal discharge; had had diphtheria 5 days previously, antitoxin 7th day	R
145	3131	♀	9	2	V.S	++	R½ removed; L completely removed	..	..	+R	+	R remnant L 0	Moderate	.....	R
146	3158	♀	18	6	V.S.	++	S	—	—	+	+	..	Moderate	Erythromelalgia and toxic eczema	R
147	3153	♀	7	2	V.S.	++	L buried	..	+	+	+	..	Mild	Diagnosis indefinite	R
148	3976	♀	2	2	V S.	++	L buried	..	+	..	..	..	Moderate	Posterior cervical adenitis; Temp. 104 F. on 11th day; incised; Temp. continued 7 days	R
149	3074	♀	7	2	V S.	++	ML buried cryptic	+	—	+	+	..	Moderate	Normal on 9th day.....	R
150	3038	♀	3	3	V.S.	++	Buried	+	+	+	+	..	Severe	Temp. normal on 10th day; severe angina	R
151	3037	♂	3	5	V.S.	++	L cryptic	..	—	..	..	..	Severe	Albuminuria 20 days; Schick 15,000; cervical adenitis; Temp. 105 15th day, normal 26th day; two other unexplained temperature rises	R
152	3050	♀	15½	4	V.S.	+	M partly buried	..	—	—	—	..	Moderate	Right cervical adenitis developed; Temp. 106 F. on 8th day; cervical nodes observed on 9th day	R
153	3257	♂	6½	?	V.S.	..	L cryptic	+	..	..	..	..	Moderate	.....	R
154	2540	♀	3	3	V.S.	++++	L buried	+	+	++	++	L more buried than R	Severe	Profuse nasal discharge; cervical adenitis required incision; hemorrhage from external jugular vein	R

adherent to it. In some cases this adhesion is the result of a preceding inflammation; in others it seems to be a congenital or normal conformation. The description "compression on swallowing" or gagging indicates that the tonsil is compressed between the anterior pillar and the superior constrictor of the pharynx when the patient gags or swallows. This observation was thought important as offering a physical explanation of the involvement of the tonsillar lymph nodes as the result of expressing toxins or organisms from the infected tonsil into the lymph stream. Of course, it is appreciated that this condition must vary during the scarlatina, as large tonsils have been seen to be everted by the pharyngeal action on admission and subsequently on becoming smaller they are compressed. This accounts for the late appearance of lymph nodes in some cases. Our observations were made on admission or within from twenty-four hours afterward. By the anterior lymph nodes we designate the deep cervical chain into which the tonsils drain. These lymph nodes lie in the anterior triangle of the neck. The posterior

TABLE 2.—SHOWING EFFECT OF PRIOR TONSILLECTOMY AND OF ADENITIS ON RASH

	All Cases 153		Previously Tonsillectomized 16 Cases (10 complete)		Adenitis 42 Cases		Severe Rash (++++ +++) 11 Cases	
	No.	%	No.	%	No.	%	No.	%
Mild	51	33	11	66	4	9	1	9
Moderate	73	47	5	32	23	56	6	54
Severe	29	18	0	0	15	36	3	27
Deaths	11	7.2	0	0	7	17	1	9

chain lies behind the sternocleidomastoid muscle and drains the nose and nasopharnyx through the retropharyngeal glands. The minus sign indicates that no glands were palpable. The plus sign indicates small glands which are just palpable and are the size of a pea. Two plus signs indicate that the glands are larger but discreet up to the size of a lima bean, while three plus signs indicate larger confluent and inflamed masses of glands. No observation indicates no record. A minus sign under difference indicates that approximately the conditions on the two sides of the throat were the same.

The character of the case is indicated as mild, moderate or severe. Mild cases are those in which the admission temperature did not exceed 102 F. and in which there were no severe complications. Moderate cases are those which followed a classical course without severe complications, the temperature returning to normal from 103 or 104 F on the third or fourth day after admission.

The notations under complications explain themselves. In cases marked "R" the patient left the hospital about thirty days or more after the onset of the disease, or later, if there had been complications.

No attempt was made to study the bacteriology of the throat and this communication concerns itself with an attempt to value the importance of tonsil anatomy on the prognosis of scarlet fever.

When the cases are classified according to their severity, and all the cases are compared with the group that had had successful tonsillectomies prior to the onset of the scarlet fever and with the group in which adenitis was a clinical feature, and, again with the group in which the rashes were very severe, it seems fair to infer that the

TABLE 3.—GROUPING OF CASES ACCORDING TO EXTENT OF ENLARGEMENT OF LYMPH NODES

	Tonsillar Lymph Nodes +++ and ++ 46 Cases		+ Nodes 70 Cases		— Nodes 11 Cases	
	No.	%	No.	%	No.	%
Rash:						
++++	2	4	..	..	1	9
+++	2	4	4	6	1	9
++	30	66	51	73	6	55
+	11	24	11	16	3	27
—	..	..	3	4	..	
Observations	45	..	69	..	11	
Tonsil Size:						
Large	30	66	39	55	3	27
Moderate	3	7	7	10	2	17
Small	12	26	15	23	3	27
Remnant	..	..	9	12		
0	..	..	..	..	3	27
Observations	45	..	70	..	11	
Adherent:						
+	31	76	52	82	0	0
—	10	24	11	18	6	100
Observations	41	..	63	..	6	
Compression:						
+	22	71	33	66	0	0
—	9	29	17	34	7	100
Observations	31	..	50	..	7	
Character:						
Severe	13	28	11	16	1	9
Moderate	22	48	29	43	4	33
Mild	11	23	28	41	6	54
Observations	46	..	68	..	11	

previously tonsillectomized cases are most favorable. There are only mild and moderate cases among them. The occurrence of a clinical adenitis doubled the percentage of severe cases and more than doubled the percentage of deaths. A severe rash had very little effect in increasing either the severity or the number of deaths.

In Table 3 the cases are grouped according to the extent of the enlargement of the lymph nodes, and I have studied the relationship to the tonsils, the extent of rash, the size of the tonsil, tonsil adhesion and compression, and finally, in accordance with the severity of the disease. Is it not a fair conclusion that the intensity of the rash and the

tonsil size bear but slight relation to the size of the nodes, as the percentages are about the same in all groups, but that when there are no nodes, there has been less adhesion and compression of tonsils and also that there are more moderate and mild cases when the lymph nodes on admission are small or absent. Probably this occurs because there is less lymphadenitis.

TABLE 4.—Analysis of Uncomplicated and Complicated Cases

	Uncomplicated (61)		Complicated (91)		Adenitis (41)	
	No.	%	No.	%	No.	%
Tonsils:						
Large	32	52	12	47	28	73
Moderate	9	11	5	20	3	7
Small	13	21	8	30	7	18
None	7	10	1	3	0	0
Adherent:						
+	27	57	9	47	22	71
—	20	43	10	52	9	29
Buried:						
+	12	..	14	87	21	96
—	..	..	2	12	1	4
With Plica:						
+	13	..	11	..	10	..
Compression:						
+	16	45	13	62	21	100
—	19	55	8	38	0	0
Nodes:						
+	41	90	15	83	34	100
—	5	10	3	17	0	0
Character:						
Severe	4	6	12	43	13	35
Moderate	24	40	10	35	21	55
Mild	33	54	6	..	4	10
Deaths	0	..	11	..	10	..

In Table 4 I have grouped the cases as uncomplicated, complicated, and those suffering from adenitis, and I have considered the size of the tonsil, the presence of adhesions, whether or not they were buried and if so, whether they were covered by plica, or were compressed, the presence of nodes on admission, and the severity of the case. At a glance it is seen that the cases suffering from adenitis are less favorable, and that tonsillar lymphadenitis is associated with an increased severity

TABLE 5

22 asymetrical cases	6	Adenitis on more covered side or on side with remnant
16 previously tonsillectomized cases	0	Adenitis

of the illness and increased mortality. There are fewer mild cases. I have emphasized in all these tables the importance of compression and covering of the tonsils because it seems to me that the organisms or toxins are forced by actual pressure into the lymph stream from the swollen tonsils. This is just as harmful as rhythmic pressure is known to be, on a phlegmon elsewhere.

Table 5 is an analysis of the asymetrical cases and the previously tonsillectomized cases. These show no adenitis where there are no

tonsils and homolateral adenitis on the worse side in the presence of asymetrical involvement.

That the anatomy of the tonsil is not the sole element in determining the character of the body's reaction to the virus is obvious, but its importance seems equally obvious. This conclusion is strengthened more by the observation of individual cases than by the statistics. On a number of occasions I have been able by pressure or manipulation to empty the infraplical pocket or a crypt of the tonsil, and have been gratified to see the swelling of the nodes recede and the temperature rapidly fall. A tonsillectomy is an important prophylactic measure against some of the so-called complications of scarlet fever, especially those arising in the throat.

Table 6 shows the conditions which have complicated our cases. I have arranged them roughly into two groups: the septic invasions which are consequent on the conditions in the nose and throat, and those other conditions which are more or less unrelated to the scarlet fever, such as vaccination, herniotomy or diphtheria. This rough grouping seems to show that the majority of the complications follow, or are associated with, a primary tonsillar infection and a subsequent cervical lymphadenitis. The benign character of most of the previously tonsillectomized cases, and the homolateral involvement of those cases with remnants of tonsil or unilaterally compressed tonsils tend to show, what many of us already accept, that the complications are really due to a secondary invasion during scarlet fever, of the tissues from a primary focus which is frequently in the tonsils. Further proof is the obvious discoverable source of infection in cases in which the tonsils were not implicated (as in the case with arthritis consequent on bad teeth, or a case not included in these statistics, where a fatal gonococcus peritonitis developed abruptly on the fifth day, from a focus lurking in the tubes).

The cervical lymphadenitis which forms such an important part in the description of scarlet fever seems from my cases to occur only when the tonsils are infected, and to be aggravated when they are swollen and pressed on while swallowing so as to express the toxins into the lymph stream. It does not occur in clean previously tonsillectomized cases and is worse on the more badly affected side in patients having tonsils.

Is it not much more rational to regard the cervical lymphadenitis as a response to drained toxins and if it is such a response why not regard the offending phlegmon in the throat as we do other phlegmons and treat it on the same principles? Elsewhere in the body the palpable lymph nodes seem to be enlarged in proportion to the amount of eruption.

TABLE 6.—Analysis of Cases, with Complications and Results

	Tonsils Diseased	Tonsils Not Diseased	Tonsil Size			Adherent		Buried		Covered with Plica	Compressed		Anterior Nodes Large on Admission		Severe	Moderate	Mild	Total Cases
			Large	Small	Moderate	+	−	+	−		+	−	+	−				
Septic Invasion from Focus																		
Continued temperature	8	3	6	3	1	4	2	5	..	4	4	..	4	1	3	6	2	11
Tonsillitis	2	..	0	2	0	1	1	1	..	1	1	..	2	..	..	2	..	2
Peritonsillar abscess	4	..	3	..	..	3	1	1	..	2	3	..	4	..	..	2	2	4
Adenitis	41	1	28	7	3	29	9	21	..	10	21	9	34	..	13	21	4	38
Nasal discharge (profuse)	6	..	5	1	1	5	2	4	..	2	5	1	6	..	6	..	1	7
Ethmoiditis	1	..	1	..	..	..	..	..	..	..	1	..	..	1	1	..	..	1
Otitis media, purulent	12	..	9	3	..	6	4	8	..	4	9	..	12	..	7	5	..	12
Mastoiditis	2	..	2	..	..	..	1	2	..	2	1	..	2	..	2	..	..	2
Arthritis	4	1	2	2	..	1	1	4	..	1	3	1	4	..	1	3	..	4
Cardiac involvement	8	..	5	1	2	5	1	5	..	2	4	2	6	1	2	4	2	8
Herpes (lateral labial)	1	3	1	1	1	..	2	1	..	..	1	1	2	..	..	..	3	3
Meningitis	1	..	1	..	..	1	..	91	1	..	..	..	1	..	1	..	..	1
Nephritis	2	1	2	..	1	1	..	2	..	..	1	2	2	..	2	1	..	3
Bronchitis	1	..	..	1	..	..	..	1	..	..	..	..	1	..	..	1	..	1
Pneumonia	5	..	4	..	1	5	..	3	..	2	2	..	5	..	4	..	..	4
Toxic eczema	1	..	..	1	..	..	1	..	..	..	..	1	1	.	..	1	..	1
Coincident Unrelated Infections																		
Diphtheria	10	1	9	..	1	5	1	5	..	3	5	..	9	..	4	3	3	10
Measles	1	..	..	1	..	..	..	1	..	1	1	..	1	..	1	.	..	1
Chicken pox	5	..	4	1	..	3	1	4	..	..	2	..	5	..	1	3	1	5
Surgical Infection: Foot	1	1	1	..	1	1	..	1	..	1	1	1	1	1	..	1	..	1
Surgical Infection: Hand	..	1	..	..	1	..	1	1	..	..	..	1	1	..	1	2	..	3
Surgical Infection: Hernia	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1
Surgical Infection: Empyema	..	1	1	..	..	1	..	..	..	..	..	1	1	..	..	1	..	1
Vaccinia severe	2	1	2	1	.	1	2	1	..	..	1	1	3	..	.	1	2	3

Why should the lymphadenitis be regarded as an integral part of scarlet fever rather than as the result of infection or inflammation in the drained area? If this view is correct, there should be more painstaking observation of the throat in scarlet fever.

I now come to a part of my study which I approach with considerable diffidence because of the fewness of the cases with which I have to deal, namely, the cases in which surgical measures were employed. Nevertheless, the narration of the individual cases gives me an opportunity to stress the importance of the anatomy of the tonsils in connection with the invasion of the body by pus organisms or their toxins during scarlet fever.

REPORT OF CASES

CASE 1.—G. Z., nurse, whose tonsillar glands were not enlarged on admission but whose small tonsils were almost covered by plica. Her temperature was rising, her nodes were enlarging and on the second day she suffered from considerable pain in the neck and throat.

Under procain anesthesia, a small horizontal incision was made in the plica which released the pressure on the tonsils. In twenty-four hours her temperature had fallen to normal, her lymph node enlargement had subsided and her case remained a mild one.

CASE 2.—A. H.; the tonsils were buried, covered with plica, adherent and with large glands. This child had a similar operation but due to the manipulation, the temperature rose for a day, the nodes, however, speedily subsided, and the patient made an uneventful convalescence.

CASE 3.—D. R. on admission had tonsils completely covered with plica, with large anterior nodes. She had a persistently high temperature, with large nodes. On the fifth day the plica was incised, and the temperature fell from 105 to 103 F. and remained at that point for three days. The general condition of the patient improved. Permission to perform a tonsillectomy was refused. The tonsils sloughed out from their beds and the patient died on the twentieth day of her disease.

Another case which enforces the importance of the anatomic structure of the tonsils was one of scarlet fever which may have been contracted in the hospital and of which we have a careful observation preceding the onset of the disease. This patient entered with no rash and no desquamation after an illness of five days. The tongue was clean and red, with prominent papillae. The throat was congested, the tonsils were adherent, especially the right. There was a mild adenitis, more marked on the right than on the left. On the fifth day her temperature went up suddenly to 105 F. Her nodes became very much more swollen and her condition serious. Operative intervention for her adherent covered tonsils was refused by her father She died on the fifteenth day of her illness, twenty days after admission.

Five cases in my series were tonsillectomized.

CASE 4.—A. G. had tonsils compressed on swallowing; they were not everted; they were buried and adherent; there were large lymph nodes. She

had a left otitis media purulenta and a facial paralysis on admission. Her mastoid was tender, there was a profuse mucopurulent discharge from her ears. Her temperature ranged up to 103.8 F., with enlarging nodes the second week of admission. She was too ill for a mastoid operation. She had been ill fourteen days, and on the twenty-seventh day of her illness, the thirteenth day after admission, her tonsils were enucleated under ether. Three days later her temperature fell to 101 F. and continued to fall; the swelling of the nodes subsided; her mastoid was then opened up and after a subsequent attack of chickenpox and diphtheria, she was discharged, after having been in the hospital eleven weeks and two days.

This patient was desperately ill and was one of five similar cases in the hospital at the time, little children with buried tonsils, enlarged lymph nodes and very marked toxemia. She was the only one who was tonsillectomized, and the only one who recovered. The operation was offered to the parents of two of the others and refused.

CASE 5.—L. W. entered as a mild case; both tonsils were covered with plica. She suffered considerable pain from the swelling of her tonsils and seven days later her temperature had risen to 103 F. and her pulse to 120. Her lymph nodes were enlarging, and there was considerable peritonsillar infiltration and the question of peritonsillar abscess was discussed. She had seen the remarkable results in the preceding case and requested tonsillectomy, which was done by Dr. Crawford, under local anesthesia. Her temperature was lower the next day and soon fell to normal.

CASE 6.—M. H. was admitted March 22 with tonsils buried, adherent and compressed on swallowing. There were large lymph nodes. She was a mild case until April 2 when she had a rising temperature with a marked cervical adenitis. Her nodes continued large until May 17 when tonsillectomy and adenoidectomy were performed. This was followed by rise of temperature and increase in size of the lymph nodes the following day. These symptoms subsided on the next day. The nodes continued to subside and the inflammation remained in one node which broke down and was incised, after which the patient recovered.

CASE 7.—D. Mc. also had large buried tonsils, a brawny infiltrated cervical adenitis, and a migrating erysipelas. The tonsils were excised; there was a prompt subsidence of the lymphadenitis. This patient recovered.

CASE 8.—The case of L. J. is instructive from many standpoints. She was admitted with an intense erythematous rash and a red throat. Her tonsils were so large on admission that they were everted and not compressed. Her lymph nodes were large and tender. April 15, six days after admission, and on the eighth day of her illness, her temperature had subsided to 101 F.; her tonsils had shrunken so as to be covered by the anterior pillar. The temperature now rose to 101.6 and 102.2 F. on subsequent days and her lymph nodes became enlarged, tender and infiltrated. Her tonsils and adenoids were excised under local anesthesia. In five days her temperature was normal and continued normal for ten days. Suddenly she developed a severe nephritis and convulsions, and finally consolidation at her left base.

This case is especially interesting because her twin sister as well as another sister were in the hospital at the same time and all suffered from nephritis as a complication and had prolonged severe illnesses. This may have been due to a similar invading organism, or a similarity in biologic reaction on the part of the body.

CONCLUSION

Firstly, my statistics and the cases narrated lend support to the view that the inflamed tonsil is a focus of infection and that the cervical lymph nodes enlarge from it in scarlet fever just as they might enlarge in the absence of scarlet fever. The inflamed tonsil becomes a phlegmon on the wall of the pharynx.

Secondly, rhythmic swallowing movements when they compress the tonsils force toxins or organisms into the lymph stream with subsequent inflammatory reaction in the adjacent lymph nodes.

Thirdly, prophylactic removal of the tonsil when buried or covered with plica or incision of the plica so as to uncover the tonsil prevents some of the severe complications arising from this source.

Fourthly, painstaking observation of the tonsil during scarlet fever is helpful, and leads to the view that the infection is conditioned by the anatomical relations.

Fifthly, in certain selected cases tonsillectomy may be performed with benefit during scarlet fever.

SOME OBSERVATIONS ON THE SO-CALLED INANITION TEMPERATURE OF THE NEW-BORN *

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For some years it has been recognized that in a certain percentage of infants there occurs in the first few days of life a rise in temperature. This fever has not been accounted for satisfactorily, so far, by any explanation which we regard as adequate to explain temperatures at other periods of life. This rise of temperature has been thought to occur with peculiar frequency at the time when the weight is at the lowest point and also in those infants whose weight loss is most marked. It, therefore, is most commonly seen on the third, fourth and fifth day. It is practically always transient in character and is accompanied by almost no disturbance of the general clinical findings. Consequently, it offers an example of a febrile condition apparently very simple in nature. It was thought that a very careful clinical study of this condition might throw some light on some of the immediate factors which cause rise of temperature.

Among the older writers Eröss,[1] in 1886, spoke of a transient fever in the new-born which fell spontaneously in some of the cases. He has regarded this fever in most cases as being due to a gastro-enteritis or, perhaps, pus at the umbilicus. This was the first mention of such a fever that we could find. The article of Sommer makes no mention of such a fever. In 1893, Herrick [2] reported a case in which the temperature rose to 106.5 F. eighteen hours after the delivery of the child. This was unaccompanied by any symptoms, and there were no signs of abrasion or inflammation. The temperature fell to 101 F. within twenty-four hours, and never afterward was above 100 F. Herrick explains this condition as probably being due to some disturbance of the cervical cord caused, possibly, by traction on the head since the delivery was with forceps. Although this temperature occurred so early, it is quite possible that this was a case of so-called inanition temperature of the new-born.

The first article dealing adequately with the subject, and the one from which this type of fever derived the name by which it is usually designated, is that published by Holt [3] in 1895. Holt uses the term "inanition fever" to describe a fever usually seen in the first four or

* Received for publication, Jan. 10, 1921.

1. Eröss: Jahrb. f. Kinderh. **24**:189, 1886; **32**:68, 1891.
2. Herrick: Arch. Pediat. **10**:337, 1893.
3. Holt: Arch. Pediat. **12**:560, 1895.

five days of life, which is independent of any local or general diseases but apparently is due to starvation. He states that this condition was first described by J. W. McLane, of New York, in 1890, before one of the medical societies of that city, at which time he presented a case. Holt reports McLane's case and then one of his own. Holt states that from observation of the new-born at the Sloane Maternity hospital it was not uncommon to find cases which in the first five days of life showed temperatures up to 102 or 104 F., accompanied by no evidence of local disease, and ceasing in nursing infants with the establishment of the free secretion of milk. This condition was observed in twenty of 200 infants at the Nursery and Child's Hospital and of 135 of 500 infants at the Sloane Maternity. The maximum loss of weight was 28 ounces. In many it was not over 20 ounces, in contrast to the average loss of weight in a healthy infant, which was 10 ounces. As a rule, the infant began to gain in weight as soon as the temperature remained at the normal point, but not until then. Holt states that he has seen this condition occasionally in older infants when they were nursing dry breasts or when fluids (food and water) had been withheld. In the discussion of Holt's paper, Christopher states that he thinks the fever is due to retention of uric acid and urates and is relieved by their excretion. Tuley,[4] in the same year, speaks of "starvation temperature," which, he says, was called to his attention by Dr. E. A. Tucker when an intern at the Sloane Maternity.

Crandall,[5] writing on inanition fever of the new-born, reports three types of cases. He thinks the evidence is in favor of the condition being due to inanition, though absolute proof is lacking. He states that in the Sloane Maternity Hospital in New York, out of 500 consecutive cases, 135 suffered from fever during the first five days of life, this not being due to septic or organic causes. In the children's hospital, twenty out of 200 showed the same symptoms.

For many years following this we have been able to find no mention of inanition temperature of the new-born, except in textbooks, such as the article in Holt's Diseases of Infancy and Childhood.

Lachs,[6] although taking up the question of the temperature of the new-born, makes no mention of the condition. Zmudzinski,[7] in 1910, in a study of the temperature and weight conditions of the new-born, states that in seventy-four of 1,665 nurslings there was a temperature increase over 37.7 C., which could not be accounted for by any recognizable disease. He divides these cases into two classes: Class 1, in which there was a single rise of temperature—consisted of fifty-four cases. Of these, twenty-eight had temperatures between 37.8 and

4. Tuley: Arch. Pediat. **12**:580, 1895.
5. Crandall: Arch. Pediat. **16**:174, 1899.
6. Lachs: Samml. klin. Vortr. (Gyn.), No. 307, 1901.
7. Zmudzinski: Inaug. Dissert., Berlin, 1910.

38 C. Twenty-one had between 38.1 and 39 C., and five had between 39.1 and 40 C. Class 2 consisted of twenty cases which showed more than one rise in temperature. Of these, two were between 37.8 and 38 C.; twelve between 38.1 and 39 C.; five between 39.1 and 40 C., and one over 40 C. Of all these cases there were only eight premature, or 10.4 per cent. In no case was there any great involvement of the general findings. Although stationary weight or loss at the time of temperature increase was recognized, he was not able to prove a connection between this condition and the rise in temperature or its unknown cause. He quotes Bedar as recognizing this condition and as stating that slight chilling or indigestion are possible causes. Of the seventy-four cases observed by him, forty-eight occurred in the first seven days of life. This suggests that there is some connection between this condition and the food or colostrum. It is altogether likely, however, that the colostrum cannot be regarded as the cause of the condition. Umbilical infections, too, are to be considered, as Runge has suggested. This, however, can scarcely be regarded as a cause of the rise of temperature in these cases.

It is not impossible that this condition may be combined either with the nature of the birth or with fever in the mother. Abnormal labors are noted in fifteen of the seventy-four cases, and in thirteen instances the mother had fever. In five other instances there were minor conditions in the infant, such as cephalhematoma, caput succedaneum, granuloma at the umbilicus, and a syphilis exanthem following a temperature of several days' duration.

The real renewal of the discussion of this subject came with a paper of von Reuss [8] in 1912. He speaks of the condition as the "fieber ohne Befund" and reports twenty-four cases. This article enters into an exhaustive discussion of the possibilities of the causes of this fever He dismisses the idea that it is of a septic nature. He also thinks that he is able to exclude in these cases the so-called ectogenous infections of the intestine since the stools in no way differ from those in the new-born without fever. While an infection with pathogenic bacteria as a cause of this transitory fever may, in all probability, be excluded, there is a possibility that it may have its origin from intestinal bacteria, their metabolic products or toxins. Against the assumption that the invasion of bacteria during the meconium period causes the fever is the time of its occurrence.

On the other hand, the vegetative changes produced by the nourishment may be of importance. Passini regards the anaerobic gas bacillus as the chief representative of the characteristic flora of the meconium. The occurrence of sugar containing milk remnants may bring about a change in the organism from the sporulating to the gas forming

8. Von Reuss: Ztschr. f. Kinderh. **4**:32, 1912.

asporogenous strain. Passini says that in this dyspepsia of the new-born, which may be regarded almost as physiologic, some toxic products appear to play a rôle. Against this statement of Passini is the fact that dyspepsia only occurs after the fourth day. After this time, if we may accept Passini's hypothesis, intestinal irritation may have a part, inasmuch as the increased alkaline intestinal secretion forms a medium in which putrefaction occurs which might cause the fever which would sink only when the milk fllora became predominant. Von Reuss mentions the fact that the intestinal wall of the new-born during the first days is permeable for high molecular colloid substances which can produce fever after their passage through the intestinal wall. He takes up the question of whether, in thermolabile organisms, like the new-born, external temperature and warm clothing may not have a part. His objections to this are, first, that the fever affects only certain individuals; second, that it does not affect the premature or debilitated children; third, that the temperature rise was between the second and fifth days; fourth, that after sponging, the temperature often increases. He feels very strongly that the fever is not a result of hindrance of heat radiation.

The next question to be decided was whether this fever is due to internal heat stagnation, that is, whether there is a disproportion between the chemical and physiologic radiation. The temperature rise occurs on the third or fourth day, which may be regarded as the critical period of the new-born. Up to this time, the body of the infant has acted as a reserve deposit. Water and other constituents have largely been excreted and, at this time, there begins to be stored up building and burning material together with water. The question presents itself as to whether this fever may be regarded as an inanition fever or as corresponding to the thirst fever which Erich Müller has described in sick infants and which Heim and John regard as salt fever. This latter is regarded as being due to a concentration of tissue juices. According to this theory, hydropigenous action of the salt leads to disturbances in warmth regulation, as a result of water combination and decrease in perspiration. Against this explanation for transitory fever is the fact that very excessive loss of weight may go along absolutely without fever. It is very hard to say whether the decreased intake of fluid is a cause or result of the fever increase.

At this period of life, there is tissue destruction, as is shown by the nitrogenous constituents of the urine. Such high molecular albumin accumulations can cause fever. It has been shown by Gein and Kotcharowski (Gundobin) that the urine of the new-born is more toxic than that of older infants or adults, and that the urine from the second to the fourth day of life is especially toxic. It is possible, therefore, that these products of protein decomposition or even salts may be the cause of the fever. In summing up, von Reuss states that the

various factors which may be responsible for the condition are the following: (1) Change from meconium to milk flora in the bowel; (2) the irritative action of bacterial decomposition products or toxins, as well as nourishment constituents and their catabolic products as such, on the intestinal cells not used to such irritation, or the resorption products on the whole organism; (3) the presence of products of tissue destruction such as one finds in the first days of life; (4) as a result of the slight fluid intake and the water impoverishment, and from this the resulting concentration of the tissue fluids and decrease of diuresis; (5) poor development of the heat regulatory apparatus. He thinks that since this condition occurs only in certain infants, it is probable that a combination of these factors is the cause.

Heller [9] reported his findings in the same year. They are summed up in Table 1.

TABLE 1.—Weight Loss in New-Born With and Without Fever

Weight Loss in the First Days in Gm.	New-born	New born with Fever	
		Number	Per Cent.
Under 200	17	0	0.0
200–300	74	4	5.4
300–500	91	24	26.3
500–720	9	5	55.5
Total	191	33	17.0

Heller states that this table shows with certainty the intimate connection between the fever increase and the size of the weight loss of the first few days of life. He states that he was, with certainty, able to exclude factors of an infectious nature. He thinks it probable that the condition is a disturbance of the concentration of body fluids brought about by water excretion, in other words, a thirst fever. A second factor may be found in the deficiency of the warmth regulatory apparatus in these infants.

In his textbook von Reuss simply repeats in an abbreviated form his conclusions in the paper.

In 1915, Jaschke [10] took up the investigation of this fever. Among 1,000 new-born infants he found thirty-one with temperature increase. In only one was there a double rise of fever above 37.5 C. In half of his cases the temperature was over 38 C., twice it was over 39 C. Aside from these cases there were twenty-two in which the temperature rose once to 37.4 C. and eighteen in which it rose once to 37.5 C. Almost never did these temperatures come during the time of physiologic weight decrease but principally in the second week of life. Twenty of his thirty-one cases, or 64.5 per cent., showed the ideal curve with

9. Heller: Ztschr. f. Kinderh. **4**:55, 1912.
10. Jaschke: Ztschr. f. Geburtsh. u. Gynäk. **78**:119, 1915.

TABLE 2.—Analysis of Data by Groups

	Weight, Gm.	Complete Series			Normal			99.2–100 F.			100.2–102 F.			Over 102 F.		
		Cases	Per Cent. Loss		Cases	Per Cent. Loss		Cases	Per Cent. Loss		Cases	Per Cent. Loss		Cases	Per Cent. Loss	
			Variation	Average		Variation	Average		Variation	Average		Variation	Average		Variation	Average
Group 1	Under 3,000	17	2.2–11.4	6.3	15	2.2–11.4	6.5	1	.....	5.8	1	.....	4.1	0	.....	.....
Group 2	3,000–3,500	44	0–12.2	6.3	31	2.1–12.2	7.0	10	0– 7.8	5.1	2	7.5– 8.9	8.2	1	.....	9.6
Group 3	3,500–4,000	81	0.8–11.1	6.7	53	0.8– 9.6	5.3	23	3.0–11.1	6.5	5	4.0–10.0	7.1	0	.....	.....
Group 4	4,000–4,500	28	1.8–14.8	6.9	14	1.8– 9.7	6.2	9	3.8–12.8	6.9	3	4.9–11.2	8.9	2	9.2–14.8	12.0
Group 5	Over 4,500	12	3.5–17.3	8.3	3	3.5–17.3	7.2	4	4.2– 7.3	6.4	5	8.6–10.1	9.0	0	.....	.....
Total.....	.....	182	0–17.3	6.8	116	0.8–17.3	6.2	47	0–12.8	6.4	16	4.0–11.2	7.9	3	9.2–14.8	11.2
Per Cent.	.....	....	.....	.....	64.2	.....	.....	25.3	.....	.....	8.8	.....	.....	1.7		

TABLE 3.—Record of Cases With Minimum and Maximum Weight Loss of Babies With Normal Temperature in Each Weight Group

Case No.	Weight in Gm.					Per Cent. Wt. Loss	Highest Daily Temperature					Breast Milk in C.c.					Cow's Milk in C.c.					Water in C.c.					Total Fluids in C.c.					Loss by Vomiting	Stools		Day Icterus Appeared
	1	2	3	4	5		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5		Meconium	Milk	
182	2,896	2,832	2,848	2,912	2,944	2.2	98.0	98.0	98.0	98.0	98.0	0	152	232	396	408	0	0	0	0.	0	72	84	72	48	72	96	184	388	592	736	0	5	7	0
183	2,128	2,064	1,984	1,888	1,920	11.4	97.4	97.6	97.6	97.4	98.2	0	16	56	184	308	0	0	0	0	0	64	32	96	56	56	72	236	304	444	480	0	4	10	3
139	3,248	3,184	3,168	3,176	3,248	2.1	99.0	98.6	98.6	98.6	98.6	0	72	356	536	560	0	0	0	0	0	96	112	32	56	176	64	48	152	240	364	0	4	3	3
144	3,136	3,088	2,880	2,832	2,752	12.2	96.0	98.4	98.8	98.4	98.4	16	32	96	104	32	0	0	0	28	0	12	40	96	240	304	28	72	192	372	336	40	0	11	0
82	3,904	3,872	3,904	3,984	3,968	0.8	98.2	98.0	99.0	98.0	98.6	0	164	352	492	644	0	0	0	0	0	80	32	32	48	24	80	196	384	540	668	0	4	18	0
47	3,832	3,648	3,536	3,464	3,544	9.6	97.0	98.4	98.0	98.0	98.0	0	0	176	224	336	0	0	0	0	0	48	168	144	176	80	48	168	320	400	416	96	5	3	0*
8	4,448	4,384	4,368	4,464	4,528	1.8	98.4	98.6	98.0	98.2	98.0	0	48	392	552	536	0	0	0	0	0	208	128	16	24	72	208	176	408	576	608	32	2	5	0
7	4,184	4,016	3,888	3,776	3,888	9.7	98.4	98.6	98.4	98.2	98.8	0	56	240	348	380	0	0	0	0	0	48	0	0	0	0	48	56	240	348	380	8	5	6	—
167	4,960	4,864	4,784	4,792	4,896	3.5	97.6	99.0	98.2	98.4	98.0	88	120	300	520	564	0	0	0	0	0	16	84	144	8	16	104	204	444	528	580	0	2	1	0
162	4,668	4,064	3,856	3,920	3,952	17.3	97.0	98.2	98.6	98.6	98.0	0	8	80	312	384	0	0	0	0	0	24	140	72	32	40	24	148	152	344	424	4	6	10	0

* Congenital eye defect.

characteristic weight increase on abundant nourishment. Two of these cases had temperatures of 40 and 40.2 C., respectively. The physiologic weight decrease amounted twice to over 300 gm., five times to over 350 gm. In the majority of all cases there was not to be recognized any connection with a condition of hunger or thirst. In forty-six of the 1,000 cases, outspoken hunger weight curves were present with the characteristic normal temperature. Of the thirty-one cases in which fever was present, in only nine did the temperature increase correspond with the lowest point of weight decrease. Therefore, he thinks that from his case one cannot conclude that weight decrease stood in direct connection with the temperature. It is the custom in their clinic to put the children to the breast a few hours after birth and to pump out the breast after nursing so that on the first day the child gets on an average from 20 to 50 gm. colostrum, and one the second day from 60 to 100 gm.

In order to test out how the children would act under other conditions, he gave fifty-six infants the following ration: For the first twenty-four hours the children were given nothing, neither breast nor water. After this they were given what they could get from the breast, whether the supply was low or not, and nothing was done to combat the undernourishment during this time. Of these fifty-six infants, ten, or 18 per cent., showed a temperature of 37.5 C. or over. This corresponds almost exactly with the figures of Heller, which amount to 17 per cent. In six of these ten cases, the temperature was over 38 C. and in two it was 39 C. In half the cases, the maximum temperature was coincident with the maximum weight loss. In general, the height of the temperature and the size of the weight loss ran parallel. Jaschke concludes from this that the frequency of the fever probably increases with the degree of the weight decrease. He prefers the term "transitory" to "hunger fever" because, he thinks, that without doubt hunger and thirst do not play the chief rôle in the causation of the temperature increase, and he agrees with von Reuss in the connection between the milk flora in the intestine and the temperature increase. He believes that hunger or thirst act in no other manner than as predisposing causes.

Mayer[11] reported eleven cases among 235 new-born. He asserts that "with the fall in temperature the increase in the weight curve usually begins." The temperature maximum and the weight minimum fall, then, as a rule, on the same day or are separated by only one day. The temperature maximum usually occurs on the third or fourth day, never later than the sixth. The children affected are strong and healthy. He explains the condition in the following way. As a result of water impoverishment, there may be a decrease in heat radiation

11. Mayer: Med. Klin. **11**:933, 1915.

TABLE 4.—Records of Cases With Temperature Between 99.2 and 100 F., Inclusive

Case No.	Weight in Gm.					Per Cent. Wt. Loss	Highest Daily Temperature					Breast Milk in C.c.					Cow's Milk in C.c.					Water in C.c.					Total Fluids in C.c.					Loss by Vomiting	Stools		Day Icterus Appeared
	1	2	3	4	5		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5		Meconium	Milk	
181	2,768	2,656	2,608	2,632	2,608	5.8	98.2	98.2	99.6	98.2	97.4	0	48	180	280	340	0	0	0	0	0	44	48	36	56	88	44	96	216	336	428	68	5	8	0
118	3,344	3,152	3,168	3,152	3,168	5.7	98.0	98.0	98.2	99.2	98.6	0	20	80	132	96	0	0	72	80	140	32	56	104	80	136	32	76	256	292	372	24	6	3	2
123	3,120	3,072	3,008	3,040	3,136	3.5	98.0	98.4	98.6	99.2	98.0	0	48	164	360	464	0	0	0	0	0	16	24	0	48	40	16	72	164	408	504	0	4	10	0
124	3,040	2,944	2,800	2,816	2,912	7.8	97.4	97.8	99.4	98.4	98.0	0	8	12	84	92	0	0	52	136	148	0	8	0	8	16	0	16	64	228	258	8	3	3	4
126	3,384	3,184	3,152	3,160	3,184	6.8	96.0	98.0	99.6	98.0	97.4	0	48	84	252	280	0	0	0	0	0	16	56	8	16	32	16	104	92	268	312	0	3	5	3
133	3,200	3,264	3,264	3,264	3,344	0.0	98.0	98.0	98.0	99.6	98.6	4	80	280	488	440	0	0	0	0	0	32	68	16	48	16	36	148	296	536	456	0	6	6	0
135	3,200	3,056	3,072	3,168	3,200	4.0	99.2	97.8	98.2	98.0	98.0	24	48	196	336	432	0	0	0	0	0	96	104	16	8	80	120	152	212	344	512	0	2	10	0
146	3,008	2,864	2,816	2,864	2,896	6.3	99.6	98.6	99.2	98.0	98.0	0	96	232	372	440	0	0	0	0	0	72	80	84	152	80	72	176	316	524	520	0	15	8	5
147	3,280	3,168	3,072	3,048	3,056	7.0	98.2	99.0	98.8	99.2	98.6	8	60	120	212	384	0	0	0	0	0	68	128	128	112	64	76	188	248	324	448	0	10	8	3
148	3,328	3,232	3,120	3,104	3,168	6.7	96.0	97.4	98.0	98.6	99.4	0	32	92	296	352	0	0	0	0	0	48	84	128	108	40	48	116	220	404	392	0	9	8	14
154	3,488	3,488	3,376	3,344	3,376	4.1	97.0	98.4	100.0	99.6	98.0	48	152	168	296	360	0	0	0	0	0	40	64	128	96	48	88	216	296	392	408	56	3	4	4
29	3,888	3,704	3,712	3,776	3,808	4.7	98.6	98.6	99.0	99.8	98.4	0	92	324	512	496	0	0	0	0	0	24	8	48	32	80	24	100	372	544	576	0	3	11	0
32	3,504	3,280	3,248	3,280	3,328	7.3	99.2	98.2	98.4	98.0	100.0	8	8	112	180	256	0	0	0	0	0	48	88	120	16	96	56	96	232	196	352	0	5	4	0
33	3,712	3,536	3,472	3,552	3,584	6.4	97.0	99.2	98.4	98.0	98.6	0	88	272	416	584	0	0	0	0	0	24	16	40	128	32	24	104	312	544	616	80	4	13	0
35	3,920	3,696	3,488	3,500	3,696	11.1	98.8	98.6	99.4	98.6	98.8	16	60	64	200	420	0	0	72	76	24	24	32	128	104	72	40	92	264	380	516	48	6	5	0*
41	3,664	3,520	3,344	3,440	3,504	8.7	98.0	99.4	98.0	99.2	98.0	0	16	172	324	472	0	0	0	0	0	0	32	32	32	16	0	48	204	356	488	0	2	3	0
42	3,568	3,440	3,360	3,488	3,520	5.8	98.2	99.0	98.0	98.2	99.4	0	12	192	368	352	0	0	0	0	0	16	20	8	0	16	16	32	200	368	368	0	7	2	0
44	3,968	3,824	3,712	3,824	3,824	6.4	99.6	98.6	98.2	98.6	97.8	0	8	160	292	444	0	0	0	0	0	8	40	48	16	0	8	48	208	308	444	0	3	6	0
51	3,888	3,872	3,680	3,632	3,696	6.5	97.0	99.0	99.4	98.6	98.4	0	32	56	340	380	0	0	0	0	0	12	96	96	48	80	12	128	152	388	460	0	1	12	0†
58	3,984	3,584	3,784	3,808	3,840	10.3	99.0	99.4	98.4	98.4	98.4	0	0	40	304	320	0	0	0	0	0	32	88	80	16	48	32	88	120	320	368	0	1	8	4
59	3,776	3,648	3,600	3,664	3,696	4.6	98.2	98.2	98.0	99.2	98.0	0	88	248	424	496	0	0	0	0	0	40	52	48	32	80	40	140	296	456	576	0	3	3	0
62	3,872	3,776	3,616	3,632	3,688	6.4	97.8	98.2	100.0	98.6	98.2	0	0	68	184	356	0	0	0	0	0	16	96	104	40	32	16	96	172	224	388	0	3	3	0
66	3,552	3,424	3,296	3,280	3,296	7.9	99.0	99.6	100.0	98.6	98.0	0	0	46	184	260	0	0	0	0	0	8	92	80	8	32	8	92	126	192	292	0	3	5	5
70	3,936	3,776	3,808	3,888	3,936	4.0	98.2	98.6	97.8	98.0	99.2	0	64	200	336	456	0	0	0	0	0	112	80	48	16	16	112	144	248	352	472	0	5	9	0
78	3,960	3,848	3,840	3,872	3,936	3.0	99.4	98.0	99.0	98.0	98.4	0	0	104	280	376	0	0	0	0	0	16	84	48	16	48	16	84	152	296	424	0	2	5	0
80	3,552	3,456	3,376	3,360	3,496	5.4	98.6	99.4	98.2	98.6	98.6	0	108	348	472	480	0	0	0	0	0	92	56	36	16	56	92	164	384	488	536	0	5	15	—
85	3,808	3,776	3,632	3,500	3,712	8.1	98.0	98.2	97.4	99.4	98.0	0	72	172	488	424	0	0	0	0	0	56	88	32	16	32	56	160	204	504	456	8	3	7	0
89	3,924	3,760	3,712	3,776	3,824	5.4	98.6	97.6	99.8	98.2	98.2	8	28	184	316	424	0	0	0	0	0	28	60	0	0	0	36	88	184	316	424	4	5	9	0
93	3,776	3,712	3,600	3,584	3,568	5.5	98.2	98.0	99.0	99.2	98.0	12	176	284	276	348	0	0	0	0	0	96	72	56	16	16	108	248	340	292	364	80	7	3	0
100	3,792	3,584	3,520	3,520	3,632	7.1	99.0	99.6	99.4	99.4	98.8	0	72	164	264	388	0	0	0	0	0	28	160	208	104	80	28	232	372	368	468	80	9	7	0
101	3,552	3,360	3,248	3,216	3,264	9.4	98.2	98.8	99.4	99.0	98.6	0	56	204	216	348	0	0	0	0	0	72	184	96	112	128	72	240	300	328	476	32	8	1	0
102	3,696	3,584	3,504	3,520	3,568	5.2	98.0	98.6	99.6	98.6	98.0	16	64	312	436	400	0	0	0	0	0	64	56	96	32	48	80	120	408	468	448	0	5	8	3
104	3,632	3,520	3,464	3,456	3,520	4.8	97.4	98.8	98.6	98.4	99.4	60	180	220	364	352	0	0	0	0	0	64	72	80	80	48	124	252	300	444	400	16	7	4	0
108	3,888	3,712	3,632	3,720	3,632	6.5	98.0	98.2	98.2	99.4	99.0	8	56	128	340	432	0	0	0	0	0	48	88	96	64	80	56	144	224	404	512	0	3	5	3—
185	4,056	3,968	3,552	3,568	3,536	12.8	97.0	99.2	99.0	98.0	98.4	16	76	172	320	180	0	0	0	0	48	80	96	88	48	64	96	172	260	368	292	0	1	0	0
11	4,320	4,256	4,110	4,192	4,256	4.8	....	98.6	100.0	98.4	99.0	0	80	284	424	48	0	0	0	0	0	32	96	40	16	8	32	176	324	440	496	0	2	6	0
12	4,304	4,160	4,016	4,048	4,096	6.7	98.0	99.2	98.0	98.0	98.2	0	16	272	312	424	0	0	0	0	0	96	144	24	32	0	96	160	296	344	424	0	5	4	0
17	4,064	3,920	3,776	3,712	3,776	8.6	98.4	98.4	98.6	99.4	98.2	0	16	108	212	312	0	0	0	0	0	48	64	16	0	0	48	80	124	212	312	0	7	5	0
19	4,160	3,952	3,856	3,912	3,984	7.2	97.8	99.0	99.0	99.6	99.4	0	92	208	364	420	0	0	0	0	0	120	92	80	16	96	120	184	288	380	516	72	0	6	3
21	4,012	3,936	3,856	3,904	3,856	3.8	98.0	97.6	99.8	98.6	99.0	24	128	332	416	460	0	0	0	0	0	72	128	200	112	208	96	256	532	528	668	0	1	11	0
23	4,064	3,936	3,840	3,856	3,888	5.5	97.8	98.6	99.0	99.2	98.6	4	168	380	400	572	0	0	0	0	0	152	48	72	32	84	156	216	452	432	656	0	4	5	0
27	4,432	4,128	4,080	4,128	4,216	8.0	98.0	99.0	99.4	98.4	98.4	24	168	228	376	376	0	0	0	0	0	72	72	72	96	80	96	240	308	472	456	0	5	2	8
28	4,208	4,024	3,960	3,968	4,008	5.9	96.0	99.2	98.6	98.4	98.4	0	32	188	352	276	0	0	0	0	0	16	92	80	64	96	16	124	268	416	372	0	2	8	0
156	4,588	4,544	4,432	4,392	4,432	4.2	98.6	99.6	98.6	99.4	98.4	0	16	92	164	244	0	0	0	0	0	0	56	72	48	16	0	72	164	212	260	0	3	3	2
158	4,784	4,672	4,448	4,480	4,544	7.0	97.0	99.2	98.2	98.0	98.2	0	16	88	296	504	0	0	0	0	0	48	144	32	32	48	48	160	120	328	552	0	3	6	0
163	5,492	5,296	5,120	5,104	5,136	7.1	99.4	98.2	100.0	98.6	98.4	0	12	24	132	112	0	0	0	0	88	96	72	48	136	176	96	84	72	268	376	0	4	3	0‡
164	4,864	4,672	4,528	4,528	4,496	7.3	99.2	99.0	98.6	99.4	99.0	0	8	152	264	320	0	0	0	0	0	60	104	72	16	64	60	112	224	280	384	72	2	3	0

* Metrorrhagia. † Enlarged thyroids. ‡ Microcephalus.

from the skin, with a resulting disturbance of heat regulation. This water impoverishment may result in diminution of urinary excretion with a result that pyrogenic metabolic products are stored up in the organism, which may disturb the normal process of removal of poisons from the body. As a result of deficient protein metabolism, liver function, etc., the new-born may already be in a "condition of light intoxication," as the toxic urine suggests. He also takes into consideration the question of the increased permeability of the intestinal wall at this period of life, and thinks it possible that since colostrum is the physiologic food for this period, the breast milk may act as a toxic substance.

In the discussion of Mayer's paper, Esch [12] disputes Mayer's idea of the importance of the change from colostrum to breast milk. He regards the fever of the new-born as a thirst fever. He thinks that the products of tissue destruction in the thermolabile new-born may be fever producing if they are retained and not excreted very quickly from the urine. He thinks that if we take this into consideration we must regard this fever as an autointoxication and the critical fall as produced by the washing out of these poisonous bodies from the body by the increased fluid intake. Jaschke,[13] in this same discussion, disputes the idea of Mayer and Esch and questions Heller's observations.

Jaschke repeats his views in his text-book, and also states that Langstein,[14] in whose clinic Heller made his observations, is convinced that hunger plays no etiologic rôle.

Lo Cicero [15] reviews the literature on transitory fever of the new-born and reports several cases, but adds nothing new in the way of observations. In general, he agrees with von Reuss.

Lindig,[16] after his experiments regarding the temperature course of new-born infants, is inclined to ascribe the transitory fever of the new-born as being due to the action of casein.

Heimann [17] reviews 8,441 cases of new-born infants with a yearly percentage of from 0.6 to 4.2 per cent. of inanition fever. The number of cases was much reduced after substituting a twelve hour for a twenty-four hour interval between birth and the first nursing. He believes that the fever is due to a number of different factors.

Our observations were made on 182 infants in the Maternity Ward of the Presbyterian Hospital, Chicago. This represented observations in 189 consecutive cases. Seven of the 189 were rejected because by careful physical examinations and laboratory findings we were able to

12. Esch: Monatschr. f. Geburtsh. u. Gynäk. **40**:162, 1914.
13. Jaschke: Monatschr. f. Geburtsh. u. Gynäk. **40**:162, 1914.
14. Langstein: Quoted by Jaschke.
15. Lo Cicero: La Pediatria **23**:768, 1915.
16. Lindig: Fortsch. d. Med. **37**:13, 1920.
17. Heimann: Monatschr. f. Geburtsh. u. Gynäk. **51**:27, 1920.

account for the fever that occurred by the presence of some febrile disease or condition, or because of prematurity or congenital debility which necessitated the taking of measures to conserve body heat.

The following routine was carried out: Immediately after birth the children were cleansed carefully and oiled. We have not made any observations here as to the delivery because this seemed to have no bearing whatever on the temperature course in the infant. The children were weighed immediately after birth and thereafter once a day, between 8 and 9 a. m. for a daily weight. The infants were put to the breast six hours after birth and thereafter every four hours, being nursed six times in twenty-four hours. Every child was weighed before and after each nursing to determine the amount obtained from each breast. Water was offered to these infants several times a day between feedings and the amount taken recorded. The temperature was taken every four hours. Urinations were recorded but no attempt was made to collect more than sample specimens. The number and character of the stools were carefully noted and recorded. If vomiting occurred, attempt was made to estimate the quantity. This, of course, could not be done accurately. Icterus was noted but had no effect on the temperature.

In Tables 2, 3, 4, 5 and 6 are noted (1) the weight for the first five days; (2) the percentage of greatest weight loss; (3) the highest daily temperature for five days; (4) the quantity of breast milk for five days; (5) the quantity of supplementary food, if any, for five days; (6) the quantity of water for five days; (7) the total fluids taken for five days; (8) estimated loss by vomiting; (9) the total number of stools in the first five days divided into, (*a*) meconium, and (*b*) milk stools; (10), and if icterus is present, the day on which it first occurred.

The cases were then divided into five groups according to weight. Group 1: Seventeen cases consisting of children weighing under 3,000 gm. at birth. Group 2: Forty-four cases, children weighing between 3,000 and 3,500 gm. Group 3: Eighty-one cases, children weighing between 3,500 and 4,000 gm. Group 4: Twenty-eight cases, children weighing between 4,000 and 4,500 gm. Group 5: Twelve cases, children weighing over 4,500 gm.

Table 1 shows very well the relation of the weight loss to the temperature in these cases. For the 118 normal cases, the average weight loss was 6.2 per cent.; for forty-six cases with a temperature between 99.2 and 100 F., the weight loss was 6.2 per cent.; for the sixteen cases with a temperature from 100.2 to 102 F., the average weight loss was 8.8 per cent., and for the three cases with a temperature of over 102 F., the weight loss was 11.2 per cent.

While at first glance this appears to be significant, still if we examine carefully the individual cases we find that among the cases

TABLE 5.—Records of Cases With Temperature Between 100.2 and 102 F., Inclusive

Case No.	Weight in Gm.					Per Cent. Wt. Loss	Highest Daily Temperature					Breast Milk in C.c.					Cow's Milk in C.c.					Water in C.c.					Total Fluids in C.c.					Loss by Vomiting	Stools		Day Icterus Appeared
	1	2	3	4	5		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5		Meconium	Milk	
174	2,144	2,112	2,048	2,048	2,080	4.1	99.4	99.0	101.4	99.4	98.8	0	0	0	16	0	0	0	64	128	96	48	104	0	0	0	48	104	64	144	96	4	5	6	0
132	3,216	3,136	2,960	2,928	3,024	8.9	97.0	96.8	98.8	100.2	98.8	0	0	32	104	192	0	0	76	132	100	12	120	108	180	164	12	120	216	416	456	16	3	4	0
149	3,280	3,264	3,104	3,032	3,072	7.5	98.0	98.0	102.0	100.4	99.0	0	12	68	232	240	0	0	0	0	0	8	96	112	96	96	8	108	180	328	336	56	7	1	0
31	3,896	3,696	3,632	3,712	3,840	6.7	98.0	99.0	100.2	98.6	99.2	0	48	40	184	304	0	0	64	80	80	72	0	64	80	84	72	48	168	348	468	24	4	8	0
34	3,984	3,584	3,696	3,760	3,856	10.0	99.0	99.0	101.2	98.6	99.2	0	36	60	304	376	0	0	0	0	0	32	16	56	80	40	32	52	116	384	416	0	5	4	0
36	3,872	3,632	3,526	3,648	3,744	8.0	99.0	101.6	100.6	98.0	98.0	52	100	212	276	352	0	0	68	28	28	88	136	116	52	60	140	236	396	356	440	0	6	7	0
76	3,664	3,536	3,440	3,552	3,680	5.8	100.2	100.8	98.4	98.2	98.0	0	28	232	364	456	0	0	0	0	0	80	96	24	80	16	80	124	256	444	472	0	3	8	2
90	3,816	3,696	3,664	3,752	3,808	4.0	98.2	98.0	100.6	99.6	98.2	0	0	96	264	360	0	0	0	0	0	120	88	72	48	8	120	88	168	312	368	0	8	2	0
3	4,224	4,192	4,080	4,032	4,016	4.9	97.8	98.6	99.0	100.2	98.4	0	28	32	88	72	0	0	0	0	92	0	72	32	64	140	0	100	64	152	304	0	7	4	0
22	4,272	4,048	3,840	3,808	3,936	10.8	97.8	99.8	100.8	100.8	98.2	0	0	140	240	364	0	0	0	24	0	32	100	168	192	88	32	100	308	456	452	120	8	7	0
24	4,192	3,968	3,792	3,632	3,856	11.2	97.6	98.2	101.6	99.4	98.6	0	36	136	124	248	0	0	16	96	72	8	120	60	136	120	8	156	212	356	440	48	8	7	0*
159	4,576	4,400	4,208	4,110	4,110	10.1	98.0	98.4	98.4	99.2	102.0	0	36	48	196	300	0	0	48	84	40	96	80	112	100	88	96	116	208	380	428	100	4	5	0
160	4,896	4,736	4,488	4,464	4,576	8.8	98.4	99.2	102.0	99.4	98.0	0	64	312	448	480	0	44	84	36	40	112	132	148	156	72	112	240	544	640	592	12	3	10	4
161	4,608	4,496	4,208	4,328	4,384	8.6	99.2	100.0	101.4	98.0	97.8	0	0	200	284	392	0	0	0	0	0	128	168	56	32	32	128	168	256	316	424	0	9	5	0
165	4,512	4,448	4,192	4,112	4,152	8.8	98.6	100.6	101.4	100.8	98.6	0	4	28	376	372	0	0	0	0	0	8	96	176	176	88	8	100	204	552	460	0	3	13	0†
166	4,600	4,264	4,192	4,256	4,384	8.8	98.8	98.0	100.4	98.4	98.0	0	52	384	496	776	0	0	0	0	0	104	120	88	64	64	104	172	472	560	840	0	2	6	0

* Congenital heart lesion.

† Gonorrheal ophthalmia (?).

TABLE 6.—Records of Cases With Temperature Over 102 F.

Case No.	Weight in Gm.					Per Cent. Wt. Loss	Highest Daily Temperature					Breast Milk in C.c.					Cow's Milk in C.c.					Water in C.c.					Total Fluids in C.c.					Loss by Vomiting	Stools		Day Icterus Appeared
	1	2	3	4	5		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5		Meconium	Milk	
134	3,472	3,392	2,232	3,136	3,232	9.6	97.2	97.6	98.2	102.4	99.4	0	0	16	96	160	0	0	36	140	128	0	180	96	172	176	0	180	148	408	464	36	1	8	0
13	4,304	4,128	3,856	3,664	3,728	14.8	98.2	98.4	98.4	103.2	99.4	0	24	48	96	264	0	0	0	52	40	48	112	72	100	72	48	136	120	248	376	76	5	7	0
26	4,432	4,336	4,128	4,032	4,144	9.2	97.4	99.6	103.0	100.8	99.0	32	172	312	272	432	0	0	0	0	0	60	96	168	136	64	92	268	480	408	496	104	4	5	0

with normal temperature the weight loss was as great as 17.3 per cent., which is higher than that found in any case with fever. A careful examination of the tables has failed to show any regular relationship between the quantity of fluid ingested and the occurrence of fever in any one of these groups; while there are cases here and there in which there seems to be a relation between the fever and the food ingested, there are also many cases in which the opposite is true.

A careful study of our observations fails to reveal any regular relationship between the inanition or transitory fever of the new-born and the quantity of fluid ingested, nor does it reveal any definite relationship between the percentage of weight loss and the temperature. We are, therefore, forced to the conclusion that the temperature rise in question is not regularly to be explained on the basis of dehydration, and since clinically the condition is clear cut, it would seem likely that the explanation for this fever must be made on some other basis. There could be very little doubt in the minds of any clinicians that forcing of food after this temperature has risen is accompanied by a fall so rapid that it seems more than likely that there is a definite causal relation between the taking of food and the fall of temperature.

We recently published an article [18] on the precipitin reaction for egg albumin in the urine of new-born infants. In it we suggested that we had been able to obtain positive reaction with such small quantities of egg albumin in such a large proportion of cases that it seemed that the presence of this reaction indicated an increased permeability of the intestinal mucosa for egg albumin during this period of life. More recently, one of us,[19] in testing the urine of the new-born for indican, confirmed the findings of von Reuss in regard to its frequent occurrence in the new-born, and especially in the third, fourth and fifth days of life. It is a well known fact, that within twenty-four hours after birth the contents of the intestinal canal have been invaded by bacteria as far as the pylorus. It has been suggested, that meconium is a poor medium for the growth of bacteria. This probably is due to the fact that meconium is so inspissated that it contains extremely small quantities of fluid, and that, therefore, the humidity is too low for the proper growth of bacteria. That, on the other hand, the meconium is not bactericidal is proven by the readiness with which it is invaded by bacteria. With the stimulation to digestion and the flow of the gastric and intestinal juices produced by the taking in of food, the meconium is unquestionably mixed with large quantities of fluid, and hence would be rendered a better material for the growth of bacteria. Meconium consists almost entirely of protein material, the destruction of which would most readily account for the presence of indican in such a large proportion of urines from new-born infants.

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18. Grulee and Bonar: Am. J. Dis. Child. **21**:89 (Jan.) 1921.
19. Bonar: Am. J. Dis. Child. **21**:406 (April) 1921.

With the addition of breast milk to the diet, the putrefactive processes in the intestine are reduced. It seems to us most likely that temperature elevation at this time may be ascribed to the absorption of some protein products, bacterial or otherwise, from the intestines of the new-born. That the condition does not give rise to more serious symptoms than are present may be explained by the transiency of the fever period and by the rapid change of the condition within the intestinal canal.

CLINICAL DEPARTMENT

CONGENITAL ABSENCE OF MIDDLE PORTION OF ESOPHAGUS *

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Cases of malformation of the esophagus are being reported more frequently, but the incidence is, of course, undetermined. Brennemann,[1] Cautley [2] and Shaw [3] reported cases so recently that it is unnecessary to enumerate them here. My case belongs to a somewhat rarer type.

REPORT OF CASE

History. — Baby V. H., girl, born Oct. 25, 1920, weight, 6½ pounds; four brothers living and well, one sister dead, cause not known. Parents well. The grandmother who is living has chorea. This child was two weeks premature, but delivery was normal. Had vomited all the milk immediately after every breast nursing. By the fourth day of life, the child had had only one bowel movement, a slight meconium-colored fluid following rectal irrigation.

Child was well nourished, had dark red lips, grunting respiration, normal temperature, no pyloric tumor, no peristaltic wave. Barium was suspended in breast milk and fed with a spoon. Fluoroscope showed a fairly large culdesac of the upper third of the esophagus with the lower margin perfectly rounded, with no evidence of any lumen beyond this point. Injection of barium and buttermilk into colon revealed a normal larger bowel. The patient was held in various positions in an unsuccessful attempt to precipitate food beyond the culdesac (Figs. 1 and 2).

With the object of creating a gastric fistula for feeding purposes the infant was operated on at the General Hospital, Oct. 30, 1920, by Dr. Harold Kuhn who has supplied the following operative notes:

Anesthetizing of skin and abdominal wall with novocain. Infant remained quiet all through the operation. Stomach delivered into left rectus incision and a rubber catheter was introduced into the stomach and held with fine hemp. This tube was buried after the fashion of a Witzel tube, with the opened end directed toward the pylorus. The stomach was small and contracted, about the size of an almond. No fluids were found therein. There was a very thin fringe of omentum from the greater curvature of the stomach. Liver down to one inch below umbilicus. In exposing the stomach the liver was retracted with difficulty and a small hematoma developed on the edge of the left lobe. In attempting to pass a catheter toward the cardia of the stomach complete obstruction was encountered. The abdomen was then closed with through and through sutures. Some hemorrhage escaped from the liver. This did not at the time, however, get into the peritoneal cavity but remained as a hematoma.

Following the operation, the baby lived forty-eight hours, during which time two teaspoonfuls of breast milk and warm water were fed through the fistula every hour. Toward the end there developed marked cyanosis.

* Received for publication Feb. 23, 1921.

1. Brennemann, J.: Am. J. Dis. Child. **5**:143 (Feb.) 1913.
2. Cautley, E.: Brit. J. Child. Dis. **14**:1, 1917.
3. Shaw, H. L. K.: Am. J. Dis. Child. **20**:507, 1920.

Fig. 1.—Anteroposterior view of the barium filled culdesac of the upper portion of the esophagus with the lower margin at the body of the fourth dorsal vertebra. (Roentgen-ray examination by Dr. E. H. Skinner.)

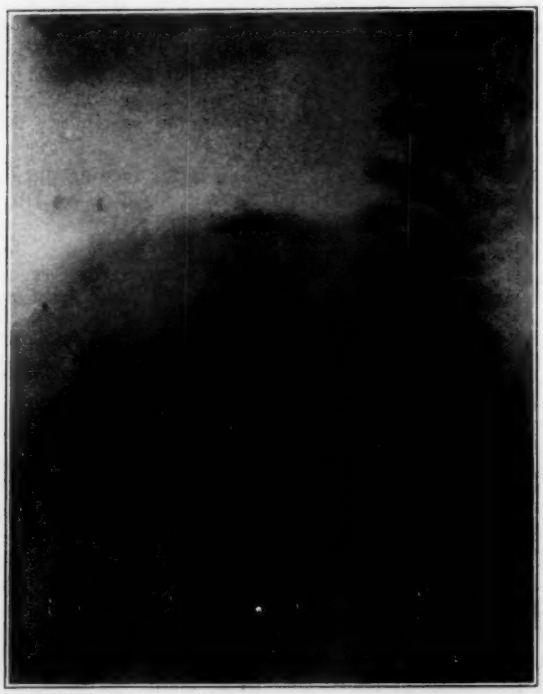

Fig. 2.—Lateral view of the barium filled esophageal culdesac showing perfectly smooth contour to the lower margin with absolutely no barium proceeding beyond the culdesac. (Roentgen-ray examination by Dr. E. H. Skinner.)

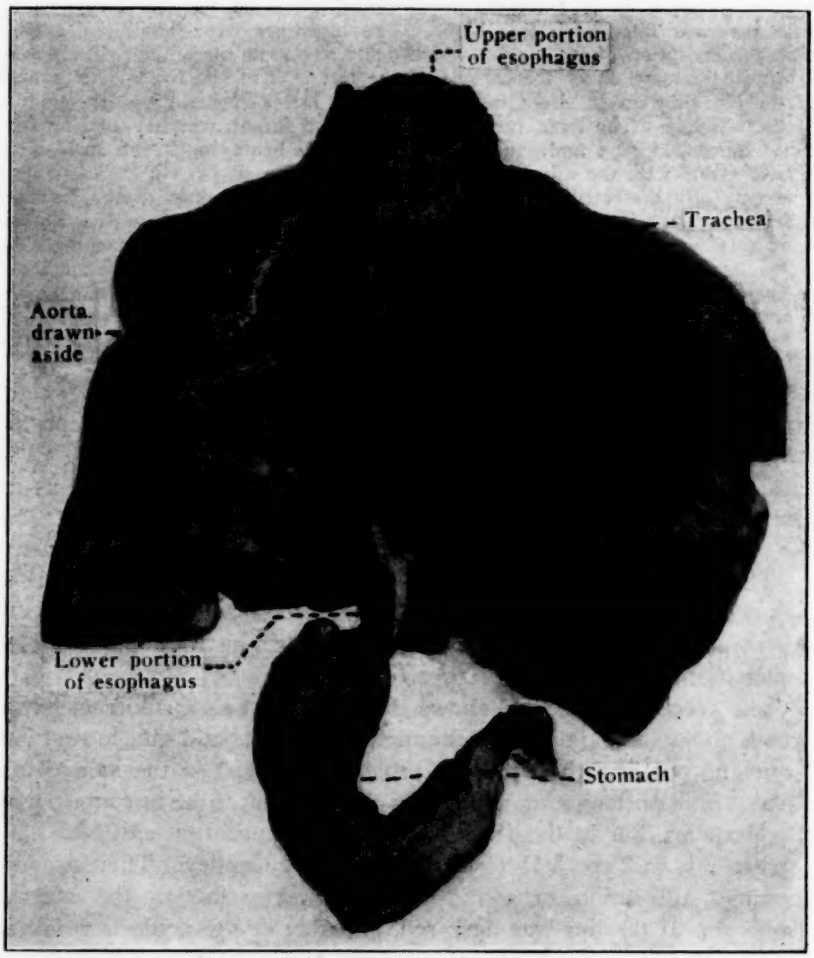

Fig. 3.—Specimen of lungs, mediastinal tissue, trachea, primary bronchi, upper and lower portions of esophagus with absence of connection between the two. The lower border of the upper portion is 0.5 cm. above the bifurcation of the trachea. Upper portion of esophagus measured laterally 1.2 cm., vertically 2 cm., its transverse lumen is 0.7 cm. Distance between upper and lower patulous portions is 4.5 cm. The lower part of the esophagus is patulous from the cardiac veins upward for a distance of 1.5 cm. and then continues in fibrous prolongations for 2 cm. The outside diameter of the lower patulous portion is 0.5 cm.

NECROPSY REPORT [4]

Body of a well-developed girl infant; postmortem rigidity present; recent operative scar just to the left of the median line extending an inch down from the costal margin, in which there was a tube containing blood. Postmortem incision made from the suprasternal notch to the symphysis pubis. Abdomen was filled with free blood. The intestines were small and empty, with the exception of the rectum which contained some meconium. There was a large hematoma under the capsule of the left lobe of the liver. The source of the bleeding was a slight laceration on the lower border about the middle of the left lobe. The liver, normal in size for an infant, was negative on section. Spleen, kidneys and pancreas negative. The heart, lungs and aorta were negative, and with the esophagus were removed in mass. When the esophagus was carefully dissected out it was found to run down only to the level of the bifurcation of the trachea, ending in the form of a pouch with a fibrous band attaching it to the trachea. Extending up from the cardiac end of the stomach was the lower portion of the esophagus with a patent lumen for about 2 cm. At this point it disappears, becoming fused with the mediastinal tissue. Stomach was small but of normal development.

Anatomic Diagnosis: Congenital absence of a portion of the esophagus.

Cautley classifies malformation of the esophagus, making eight general divisions. He cites the literature on the previously reported cases, showing the rarity therein, especially for certain types. The case here reported belongs to the seventh group and is classed as an atresia, with the upper part normal, the middle portion missing, and the lower portion reappearing above the cardiac end of the stomach.

The embryonal development of esophageal malformation has been ascribed to the nonformation of a tracheoesophageal septum. In my case a glance at the drawing will cause one to doubt such an explanation.

The specimen presented shows the upper part as a culdesac, rather broad, the middle portion obliterated or absent, and the lower part branching out into three fibrous prolongations lost in the mediastinal tissue. One prolongation appears in the drawing to be in contact with the bronchus, but in the fresh state no communication existed. This corresponds to Type VII (b) as classified by Cautley. There was no choking, suffocation or cyanosis in this case, though the mucous membrane of the lips was dark red, probably due to acidosis resulting from starvation. The lungs were free of any pneumonic process. The walls of the culdesac are thick and muscular as mentioned by Kastner [5] in his case. The stomach was not distended with air, and was smaller before the operation than afterwards.

4. Necropsy done by Dr. Watson Campbell, pathologist at the General Hospital. Specimen removed and preserved, from which the drawing was made.

5. Kastner, A. L.: Arch. Pediat. **37**:670, 1920.

PROGRESS IN PEDIATRICS

RECENT PROGRESS IN ANATOMY, PHYSIOLOGY AND PATHOLOGY OF CHILDHOOD

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ANATOMY AND CONGENITAL ANOMALIES

Growth.—There remains much uncertainty as to just what are to be regarded as normal height and weight for children of different ages. A conference for the purpose of adopting a uniform table of weight and height for children of various ages was held in 1920, and was attended by representatives of leading associations and departments of the government interested in child welfare. The findings of this body will be of much interest.

Pooler [1] reviews some factors influencing growth in infancy and childhood.

Weber [2] has made a survey of sixty-two cases of congenital heart defect in children in the Universitäts-Kinderklinik, Berlin. He states that he was unable to discover any characteristic malnutrition or stunting of growth in the children with cardiac anomalies. It was evident, however, that children with congenital cyanosis, and especially those with symptoms of congestion in various organs, were more subject to deranged nutrition than other children with congenital cardiac disease. Children with dyspnea, especially in combination with cyanosis, likewise showed evidence of malnutrition. Hypotrophy was not found to be a constant accompaniment of congenital heart defects and no constant relation was found between the two conditions. Hypotrophy was nearly always associated with the presence of other defects, so that it was thought necessary to look farther back for the destructive agency. However, in some cases, no damaging factor could be discovered other than the congenital heart defect.

An interesting example of oxycephalia and dwarf growth is reported by Isola, Butler and Fournier.[3] The patient was 21 years of age and 1.3 m. tall, totally blind, with reducible exophthalmus and horizontal nystagmus. The skull showed scattered depressions where the con-

1. Pooler, H. W.: Some Factors Influencing Growth in Infancy and Childhood. The Child, 1919, 396. Abst. Arch. Pediat. **36**:505, 1919.

2. Weber, A.: Growth and Nutrition of Children with Congenital Heart Defect, Monatschr. f. Kinderh. **18**:205, 1921.

3. Isola, A., Butler, C., and Fournier, J. L. Mussio: An. de la Fac. de méd., Montevideo **5**:255, 1920.

volutions of the brain had exerted abnormal pressure on the inner table, inducing rarefaction. At the age of 2 years, vomiting and headache were followed by progressive loss of vision. The retrospective diagnosis is that an attack of hydrocephalus injured the pituitary early in life and the dwarf growth was secondary to this.

Reuben [4] describes, with photographs, an interesting example of adiposity in a breast-fed infant. Physicians who had seen the child regarded this as a case of dispituitarism, but the cause was found to lie in the excessive fat content of the mother's milk, from 6 to 7 per cent.

Anomalies.—A notable case of situs inversus viscerum totalis is described by Lineback.[5] Karashima, in 1912, in describing a case of situs inversus totalis, gave a catalogue of such cases up to his time, something more than 200 cases. Lineback's specimen was a near-term female fetus selected from the dissecting material at the University of Chicago. No data could be found regarding its history, it weighed at the time of study, 3,000 gm. and all the general outward appearances were good. The case is interesting because of the great number of anomalies, some of them anomalies of reversion. The fully developed body was served by a heart of an early embryonic state. The real value of the case is in the positive data it offers to the problem of placing the causative factor for such anomalies. Trauma, from various sources, in late embryonic life, might explain external deformity, and some kinds of internal derangements, but Lineback finds it difficult to see its effect on reversion. Nor does he think applicable Mall's view that monsters are products from normal embryos "by influences which are to be sought in their environment." More appropriate, in his opinion, is the use of the term "cytoplasmic variation," in which it is suggested that in the germ cell there is predilection for the ultimate form of the body, which at least influences these malformations. Attention is directed, then, to an earlier, a very early period in development. The present case is thought to afford proof, as instanced by reversed anomalous aortic branches, primitive heart, reversed root of the lungs and azygos veins, and the relationship of the pancreas and the unrotated intestines, that these anomalies have their causative factor operating at a very early embryonic period.

Goldstein [6] has made a study of a monster with the olefactory lobe and most of the hind brain missing (arhinencephalia) which is

4. Reuben: Adiposity, Cerebral versus Lacteal, Arch. Pediat. **36**:636, 1919.

5. Lineback, P. E.: An Extraordinary Case of Situs Inversus Viscerum Totalis, J. A. M. A. **75**:1775 (Dec. 25) 1920.

6. Goldstein, A.: Arhinencephalia Monstrosity, Ztschr. f. Kinderh. **25**:328, 1920.

thought to confirm the assumption that pressure or traction from the amniom is responsible for such deformities, as also cyclops monstrosities.

Congenital Aneurysm.—Goehring [7] reports a case of congenital aneurysm of the posterior aortic sinus of Valsalva with an anomaly of the aortic cusps. Six cases of aneurysm of the sinus of Valsalva are recorded in the literature. In Goehring's case the aneurysm projected into the right auricle so as to produce a tricuspid insufficiency, which was the chief clinical manifestation. Sudden progressive heart failure with dyspnea, cyanosis and absence of the radial pulse during six hours before death, was probably related to the rupture of the aneurysm.

Thymus.—In 78.25 per cent. of 115 cadavers of young children examined by Canelli,[8] the thymus appeared in two lobes; in 74.77 per cent. of the cases the thymus was in the thorax. The water content of the gland seemed to be in inverse proportion to the age of the patient; while the weight after desiccation increased with the age.

Park and McClure's [9] study on the effect of thymus extirpation in the dog is most interesting in this connection. The experimental literature of thymus extirpation is reviewed with unusual care. Many earlier assumptions are shown to be unfounded.

Heart.—K. Preisich [10] gives figures for the size of the heart in infants and children from thirty-five child cadavers.

Diaphragm.—DeBuys [11] reports an anomaly of the diaphragm with a gastro-intestinal obstruction.

Intestine.—Robbin [12] reports the results of a study of the length of the large and the small intestine in young children.

Mammary Glands.—A study by Meyers [13] deals with the development of the mammary glands from their earliest appearance until the period of pregnancy.

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7. Goehring, C.: Congenital Aneurysm of the Aortic Sinus of Valsalva, J. M. Research **42**:49, 1920.

8. Canelli, A. P.: Anatomy and Pathology of the Thymus in Young Children, Pediatria, Naples **28**:753, 1920.

9. Park, E. A. and McClure, R. D.: The Results of Thymus Extirpation in the Dog with a Review of the Experimental Literature on Thymus Extirpation, Am J. Dis. Child. **18**:317, 1919.

10. Preisich, K.: Size of the Heart in Young Children, Jahrb. f. Kinderh. **92**:400, 1920.

11. DeBuys, L. R.: A Case of Anomaly of the Diaphragm with a Gastric and Intestinal Obstruction, Arch. Pediat. **36**:360, 1919.

12. Robbin, L.: The Length of the Large and the Small Intestine in Young Children, Am. J. Dis. Child. **19**:370 (May) 1920.

13. Meyers, J. A.: Studies in the Mammary Gland. VI. The Development of the Mammary Gland from Its Earliest Appearance Until Period of Pregnancy, Am. J. Dis. Child. **18**:4 (July) 1919.

Wernstedt [14] reports the findings at necropsy in two infants with congenital stridor, comparing the findings with those in other infant cadavers. In one of the two cases the lumen was abnormally narrow. The other case seemed to be an instance of functional disturbance.

Esophagus.—Morse [15] reports two cases of congenital stricture of the esophagus.

Extremities.—Nuzzi [16] tabulates the details of eighty-one cases of congenital deformity of one or both legs due to congenital absence of the tibia, including three operative cases from the Rizzoli Institute. It is advised that amputation be postponed as the growth of the child will modify the stump. The details of conservative intervention vary from case to case as there are usually other deformities. Those of the knee and ankle are generally secondary, which facilitates functional use of the limb.

A paper by Ried [17] deals with congenital malformation of the spine, and Stern [18] reports an instance of congenital absence of both clavicles. In Stern's case no other anomaly was found and the condition seemed to be one affecting only the male line of the family (seven affected), the females escaping.

An instance of congenital absence of the right femur in an uniovular twin is reported by Harrison.[19]

The association of acrocephaly with deformities of the extremities is considered by Park and Powers.[20]

Urogenital.—MacKenzie [21] reports an instance of congenital malformation in which two testes and a uterus are described as being present in the right portion of a scrotum.

Pybus [22] reports a case of congenital urethrocele, and Thompson [23] cites an instance of ectopic urinary bladder.

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14. Wernstedt, W.: The Pathological Anatomy of Congenital Stridor, Hygiea, Stockholm **82**:609, 1920.
15. Morse, J. L.: A Report of Two Cases of Congenital Stricture of the Esophagus, Am. J. Dis. Child. **19**:144 (Feb.) 1920.
16. Nuzzi, N. O.: Chi. d. Org. di Movimento **4**:164, 1920.
17. Ried, C. D.: Congenital Malformations of the Spine, New York State J. M. **19**:161, 1919.
18. Stern, W. G.: Congenital Absence of Both Clavicles, J. A. M. A. **73**: 1526 (Nov. 15) 1919.
19. Harrison, V.: Uniovular Twin with Congenital Absence of Right Femur, Virginia M. Month. **46**:54, 1919.
20. Park, E. A. and Powers, G. F.: Acrocephaly with Deformities of the Extremities, Am. J. Dis. Child. **20**:235 (Oct.) 1920.
21. MacKenzie, D. W.: Congenital Malformation: Two Testes and Uterus in Right Portion of Scrotum, Canad. M. A. J. **9**:641, 1919.
22. Pybus, F. C.: Case of Congenital Urethrocele, Brit. J. Child. Dis. **16**:87, 1919.
23. Thompson, R.: Case of Ectopic Urinary Bladder, Brit. J. Child. Dis. **16**:80, 1919.

Eye.—Sixty-four cases of hereditary congenital ptosis, which conformed to the mendelian rule for dominance, are reported by Briggs.[24] Middleton [25] tabulates the congenital conditions affecting vision found in examining a large body of troops.

PHYSIOLOGY

Nutrition.—Rich [26] reports on the direct nutritional work done in the public schools of Chicago. The increasing cost of living, sending more and more children aged 14 into the ranks of the workers, led to the discovery that 53 per cent. of the children aged 14 applying for employment certificates were from 7 to 21 per cent. below the weight of the average child of that age, that these children, when they were employed, could not carry on their normal amount of work, and that because of this they lost their jobs, became discouraged and gravitated into becoming rolling stones, thus easily falling prey to vicious and criminal companions.

The Board of Education determined to ascertain the effect that direct nutritional work would have on pre-industrial children in giving them the requisite bodily fitness for the labor they must perform.

To meet the standard of the Employment Certificate Bureau of the Chicago Public Schools the child of 14, in order to obtain a working certificate (with a few exceptions) must measure 57⅕ inches in height and weigh 80 pounds. (This applies also to children who intend going into the high school but wish to work during the vacation period).

Five public schools in both foreign and native localities were selected for the experimental work. The groups included children who were given milk, rest periods in cots, noonday lunches, and graduated physical exercise under a director (children of an "open window" school), children who were given milk but no rest period, children who were given neither milk nor rest periods, but simply instruction to be carried out at home, and combinations of these methods.

A chart was prepared to meet the needs both of the public schools and of the employment certificate bureau. It was formulated in such a way that comparative gains both in height and weight are seen at a glance. A certificate of graduation (filling the requirements of the employment bureau) was given each child, signed by the physician in charge, the nutritional worker, and the principal of the school. This was presented with the other papers (birth certificate, etc.) to the

24. Briggs, H. H.: Hereditary Congenital Ptosis; Report of Sixty-four Cases Conforming to Mendelian Rule for Dominance, Am. J. Ophth. **2**:407, 1919.

25. Middleton, A. B.: Congenital Conditions of the Eye Affecting Vision Among Troops Examined at Camp Travis, Texas, Am. J. Ophth. **2**:377, 1919.

26. Rich, K. B.: Nutritional Work in Public Schools, etc., J. A. M. A. **75**:226 (July 24) 1492 (Nov. 27) 1920.

employment certificate bureau when the child applied for its working certificate, which gave it dignity at once and caused the document to be prized highly.

It was found that the children prefer to consider the matter their own business—their first real "job" in life—and that they get coöperation at home when they feel the need of it. The nutritional worker had little effort to expend in reaching the parents, and the question is raised whether or not the children who assume the responsibility for their own improvement will not do better than those for whom the effort is made by others.

While some of the classes for whom milk was provided made good gains, those who had no milk also gained well. It seemed quite probable that, as school interest is further developed, fully as good results will be obtained without the necessity of providing milk during the school sessions. Rest periods were taken at home, and early hours for retiring were observed, this being part of the responsibility that the class members assumed.

Many physical defects were corrected, the consent of the parents being obtained, in many cases, by the children themselves.

One of the interesting points brought out in the work is that underweight previously was thought to result largely from bodily defects—diseased tonsils and teeth, adenoids, flat foot, etc.—hence, strictly a medical question. In the experiment, however, it was found that as the children gained in weight they appreciably gained in height without any medical care whatever.

One child sent to the clinic by the public employment bureau was 2 inches under height and 10 pounds under weight; in four months he gained 2⅕ inches in height and weighed 84 pounds. Others made equal gains.

The development of the work indicates that undernourished children may be placed in three groups: (1) infants and young children up to the age of 6 years; (2) children from 6 to 13 years, and (3) boys and girls from 13 through the period of high school education. The handling of the first two groups is an expensive undertaking, requiring the coöperation of the parents and many visits of a capable worker, and seems best done by organizations outside of the school system. The third group, on the contrary, is found to be controlled almost exclusively through the proper presentation of the problem to the individual student, and through his own interested effort to become physically and mentally fit. "It is largely a question of psychology, and presents a great opportunity for character building."

A re-examination recently made of two of last year's classes (1919) seems to justify the theory that boys and girls of the pre-industrial

age should be taught, primarily, by leading them to assume the responsibility of their own welfare.

The Kansas City experiment was rather different.[27] Following the influenza vacation, the children of fifty-five grade schools were weighed and measured by the classroom weight-chart method of the Child Health Organization. The prevalence of malnutrition corresponded with that found elsewhere. It was decided to take the school that showed the highest percentage of children who were more than 10 per cent. under weight by the standard table of Wood, and see what would be the effect of an intensive health service for the "malnourished" children. The school selected showed 41 per cent. of its children more than 10 per cent. underweight.

The children came from families of men earning from $50 to $200 a month, and almost entirely American. The services of an antituberculosis society were utilized, including medical and dental attention, nurse's service, open window schoolroom, shower baths, and midmorning and midafternoon lunch, followed by naps. Circumstances happened to divide the underweight pupils into two almost equal groups—112 who were given school feeding and 109, identical in respect to physical condition as nearly as could be determined, who were considered the "control group." A trained nurse gave her entire time to this demonstration group, visiting every home, carrying the recommendations of the physician, and soliciting the cooperation of the mother. The home dietary was investigated, and such instructions given the mother as conditions seemed to warrant.

The school feeding given the demonstration group was planned to supplement the home dietary, and to supply "about half (from 1,000 to 1,200 calories) of the necessary days ration." The principle of "protective feeding" as developed by McCollum was followed. Each child received from 1 to 2 pints of milk with cereal and fruit every day, and the remainder of the 1,000 to 1,200 calories was variously supplied in soups, vegetables and sandwiches. Cocoa was frequently served, with occasional treats of cookies and milk chocolate. The cost of the food averaged 6½ cents a meal (April, May, June, 1919).

After the first month, each pupil who lost weight was given an extra half pint of milk, and during the last month each child who had lost weight was given in addition to the half pint of milk, 100 calories of dates, raisins or chocolate, some of the group by this time receiving up to 1,800 calories of school feeding a day. The pupils ranged from the kindergarten to the sixth grade.

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27. Brown, M. A.: A Study of Malnutrition of School Children, J. A. M. A. **75**:27 (July 3) 1920.

No child was dropped from the group for any cause. Those who were absent for several weeks on account of infectious diseases, or who were in the hospital for tonsil and adenoid operations, gained enormously on their return to the class; but failed to make the normal gain for the whole period. One girl with badly diseased tonsils, which her parents refused to have removed, made little gain. Two failures were directly traceable to adverse home conditions; one child moved away. In yet another case, the only adequate reason for failure to gain was thought to lie in the fact that the child had too little rest and play, as she attended a religious school after public school hours. One boy would not gain; the more he ate, the thinner and stronger he became; he played on the baseball team and was apparently in the best of health; it was concluded that he was one of the "naturally thin" children one hears about.

The weight curves of the children of the feeding groups showed these features: (1) An initial rise, no matter what the medical or social condition of the child, and an apparent response of the underfed tissues of food. (2) A failure to gain in the next month, so that by the end of it half the group were receiving extra food. (3) A rise in the third month (June). Factors other than purely metabolic ones, weather, etc., may have played a part in these fluctuations.

As a result of this nine-and-a-half weeks' experiment, these children, in spite of adverse conditions and handicaps unremoved, gained 278 per cent. (normal gain 100 per cent.), or 302 per cent. gain over the "control group," and the school which had ranked lowest (fifty-fifth) in the spring, ranked third in September.

Blanton [28] writes of the mental and nervous changes caused by malnutrition among German children.

Changes in the teeth of children due to nutritional disturbances are discussed by Sinclair [29] and by Castilla.[30]

Those who assert that good health is purchaseable must take into consideration the cost of providing adequate nutrition and the limitations of income. On the basis of the prices of April, 1920, the director of a league for preventive work, in Boston, calculated the cost of providing a minimum allowance of essential nutrients for a family of five as $11.50 per week. This would allow, for the man, 3,400 calories a day, including 100 gm. protein, 1 gm. calcium, 2 gm. phosphorus, and 16 gm. iron, with an abundance of vitamins. A more liberal, safer,

28. Blanton, S.: Mental and Nervous Changes in School Children of Trier, Germany, Caused by Malnutrition, Mental Hygiene **3**:343, 1913.

29. Sinclair, J. F.: Influences of Diet Affecting Second Dentition, Penn. M. J. **12**:789, 1919.

30. Castilla, C. R.: Changes in Teeth of Children from Nutritional Derangement, Semana méd. **26**:599, 1919.

allowance is placed at $14, or 60 cents a day for each adult.[31] Since the date of this calculation there has been a very material decline in the cost of these essentials; the figures must be modified from time to time as prices return toward the prewar level.

Milk.—The "misuse of milk" in the diet of infants and young children was discussed at the meeting of the American Pediatric Society, and the view was advanced that following propaganda by popular writers many mothers are forcing milk in such large quantities on their children that it becomes a positive menace to health, particularly in the group of children between the ages of 1 and 6 years. In receiving a diet so largely composed of milk, the child is deprived of minerals that should come to it in fresh fruits, vegetables and cereals, as well as the vitamins and antiscorbutic properties of the foods. Hoobler[32] advocated limiting the amount of milk to one pint daily after the child is 15 months old.

Bergeim[33] reports upon a study of coagulation of milk in the human stomach.

Sommer and Hart[34] found that the main factor in the heat coagulation of fresh milk is the composition of the milk salts. Apparently casein requires a definite optimum calcium content for its maximum stability. The calcium content of casein is largely controlled by the magnesium, the citrates and the phosphates present.

In fresh milk there is no relation between titratable acidity and heat coagulation Acid fermentation in milk lowers the coagulating point by changing the reaction and by lowering the citric acid content. (However the titratable acidity of fresh milk samples varies so widely that it is impossible to determine the extent of acid fermentation by titration. Therefore, it is impossible to use the acidity of milk as a criterion of coagulability.)

Difference in concentration accounts partly for the difference in coagulation of fresh milk samples. Hydrogen ion concentration is not the determining factor in fresh milk coagulation. Nevertheless it is a factor in fresh milks, and in commercial milks it may become an important factor.

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31. Gillett, L. H.: A Minimum Food Allowance and a Basic Food Order, J. Home Econ. **12**:319, 1920.

32. Hoobler, R. B.: Tr. Am. Pediat. Soc., 1920.

33. Bergeim, O.: Fractional Study of Coagulation of Milk in Human Stomach, Am. J. Physiol. **48**:411, 1919.

34. Sommer, H. H. and Hart, E. B.: Heat Coagulation of Milk, J. Biol. Chem. **40**:137, 1919.

Dennis and Talbot [35] have studied the lactose, fat and protein content of woman's milk. Talbot states [36] that the caloric value of lactose is 3.7, and not 4.1, as taught.

C. U. Moore [36] writes on re-establishment and development of the breast milk flow.

From Munich it is reported that the use of surplus mother's milk in hospitals has brought the danger of adulteration with cow's milk or dilution with water, as the women furnishing such milk are paid a good price for it. Kappeller and Gottfried [37] state that if as much as 20 per cent. cow's milk or 10 per cent. water has been added, the adulteration may be detected by changes in the Umikoff reaction, in the refraction of light, and in the precipitation of casein.

Legrand [38] reports instances in which, after puppies had been drowned, the dog suckled an infant. In one case he advised utilizing the large female dog he noticed under a gypsies' wagon, when he saw that the gypsy twins he had delivered not long before were being fed on unsanitary soup. The animal served with evident delight, and the infants thrived. An instance is cited to show that lactation can be kept up for from five to six months.

Proteins.—Further evidence that the proteins contained in the commonest cereals are essentially equivalent is found in recent work by Osborne and Mendel.[39] Wheat was contrasted with barley, rye and oats in experiments in which these served as the sole sources of protein for growth, and it was concluded that the proteins of the four cereals studied are not widely different in efficiency as sources of nitrogen for growth. All, however, need a supplement to provide certain inorganic elements and fat soluble vitamin.

All the common cereals are readily digestible by man, and all have virtually the same energy value. A 100 calory portion varies only from 25 gm. for oatmeal (dry flakes) to 29 gm. for rice. A choice of one cereal rather than another is to be made on grounds of transportability, personal preference, etc., rather than on grounds of nutritional value.

It appears that nine tenths of the protein requirements of maintenance of the adult human organism may be met with protein derived

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35. Dennis, W., and Talbot, F. B.: A Study of Lactose, Fat and Protein Content of Women's Milk, Am. J. Dis. Child. **18**:93 (Aug.) 1919.

36. Moore, C. U.: Reestablishment and Development of Breast Milk, Arch. Pediat. **36**:609, 1917.

37. Kappeller and Gottfried: Adulteration of Human Milk, Münch. med. Wchnschr. **67**:813, 1920.

38. Legrand, A.: Dog's Milk for Infants, Nourrison **8**:223, 1920.

39. Osborn, T. B. and Mendel, L. B.: Nutritive Value of the Proteins of the Barley, Oat, Rye and Wheat Kernels, J. Biol. Chem. **41**:275, 1920.

from cereal grains (wheat, maize and oats), the remainder being furnished by milk or fresh fruit.[40]

Many interesting facts about the proteins of animal and plant life are given by Kennaway.[41] These giant molecules, the most complex of organic substances, occur only in association with living matter. The number of different amino-acids that enter into their formation is limited, but as there are about seventeen commonly found, the possibility for variation by combination and arrangement is practically unlimited. Why other and related amino-acids are not found in nature and do not enter into the formation of proteins is unexplained; as is the fact that proteins usually contain amino-acids with two, three, five or six carbon atoms, and four carbon amino-acids are not found. All the amino-acids of animal proteins are synthesized by plants and bacteria, and if animals have any slight capacity to synthesize amino-acids and proteins it is but very limited and of little significance as a usual source of proteins. No single amino-acid has been found in animals that is not present in plants. And in the simplest organisms that have yet been analyzed—yeast, moulds and certain bacteria—are found virtually all the known amino-acids of animal and plant proteins, with the exception of the sulphur containing cystin. Moreover, the simplest organisms now existing apparently do not contain a series of amino-acids any more primitive than that present in the higher organisms, except, perhaps, as regards the inclusion of cystin. It would seem that any struggle for existence among simple organisms selecting other series of amino-acids must have taken place in an immensely remote period, leaving the present apparently stereotyped series of utilizable amino-acids as the stable outcome. From extremely simple inorganic salts, bacteria and plants, e. g., yeast growing on a medium containing ammonium tartarate, can synthesize extremely complex protein molecules so rapidly that a new cell is formed in a few hours; the chemical processes cannot be followed.

Evidence that cystin is essential for growth in higher organisms (dogs) is presented by Lewis.[42] Under conditions of low protein intake, he finds that serum albumin with a high cystin content, is more effective in maintaining nitrogeneous equilibrium than is casein, a protein low in cystin content. On the other hand, when casein is supplemented by cystin, it becomes as efficient as serum albumen.

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40. Sherman, H. C.: Protein Requirement of Maintenance in Man and the Nutritive Efficiency of Bread Protein, J. Biol. Chem. **41**:97, 1920.

41. Kennaway, E. L.: Notes on the Evolution of Protein, Chemical News, **120**:13, 1920; Ed. J. A. M. A. **75**:744, 1920.

42. Lewis, H. B.: Metabolism of Sulphur. III. Relation Between Cystin Content of Proteins and Their Efficiency in Maintenance of Nitrogeneous Equilibrium in Dogs, J. Biol. Chem. **42**:289, 1920.

Whether ammonium salts, such as ammonium chlorid, acetate and citrate, can replace protein as a source of nitrogen for the tissues, and serve as "protein sparers," has been subjected to further study by Gessler,[43] at Heidelberg. True protein contains sulphur as well as nitrogen, and organic sulphur is quantitatively as indispensable to the organism as is organic nitrogen. When protein disintegrates, substances containing sulphur appear in the excreta, and there is a parallelism between the two elements. If ammonium salts lead to protein sparing, the loss of sulphur should be decreased correspondingly. This, Gessler finds, is not the case. Apparently, proteins built up by plants and animals, even though expensive and at times difficult to procure, are essential for supplying this element of human nutrition.

Hanke and Koessler [44] report the results of study on the recurrence of histamin in casein, blood serum, and fresh beef hypophysis. By means of a colorimetric method, it is possible to detect accurately quantities of histamin (B-imidoazolylethylamin) as small as 0.1 mg. in protein and protein containing matter. It could not be demonstrated in 40 gm. casein or in 75 c.c. human blood serum; and when added to either substance before hydrolyzing, it could be recovered quantitatively. Histamin is not found in perfectly fresh beef hypophysis. Casein contains a depressor substance that is similar to histamin pharmacologically. Peptone shock and histamin shock are not identical.

The foremost representatives of disintegration of protein in the body appear to be the amino acids. They are formed on a large scale during, for example, resolution in pneumonia. They circulate in the blood and are formed rapidly into urea, and excreted, and do not remain unconverted. With the appearance of convincing evidence that urea can be formed in tissues other than the liver, there has arisen some question whether the liver is so important in the disposition of the products of protein katabolism as had been supposed. Stadie and Van Slyke [45] have investigated this. In acute yellow atrophy, organ systems other than the liver are not prominently involved, at least histologically. Opportunity is thus afforded to test the effect of the injury to the liver on urea formation.

It is found that the rapid antolysis of liver tissue liberates amino-acids in large amounts, and they then appear in the blood to a degree not equalled in any other human condition. The urea content of the

43. Gessler, H.: Zur Frage des Wesens der Stickstoffretention bei Fütterung mit Ammoniaksalzen, Ztschr. f. Physiol. Chem. **109**:280, 1920.

44. Hanke, M. T. and Koessler, K. K.: Studies on Proteinogeneous Amins, etc., J. Biol. Chem. **43**:527, 1920.

45. Stadie, W. C., and Van Slyke, D. D.: The Effect of Acute Yellow Atrophy on Metabolism and on the Composition of the Liver, Arch. Int. Med. **25**:693 (May) 1920.

blood is not increased correspondingly. For some reason the amino-acids are not deaminated as rapidly as they should be; they circulate and are excreted as such. From these facts, it is assumed that the liver bears an important part in the deamination of amino-acids and the production of urea, and one that cannot be assumed by the rest of the body.

The use of low protein diets in the treatment of certain affections of the skin is familiar. Shamberg [46] has utilized this treatment in psoriasis, and it has been extended to other dermatoses. Blackfan,[47] most recently, has advocated the entire withdrawal of protein from the diet of children who are hypersensitive to protein and who are unable to take various foods without the development of eczema, urticaria, asthma, etc.

The reduction or complete removal of protein from a diet throws a great burden on the carbohydrate tolerance of the body and this may not always be harmless.

Michael [48] points out that this treatment in a prediabetic (adult at least) may lead to diabetes. Frequent blood sugar estimations should be made, as potential diabetes will usually manifest itself through hyperglycemia.

E. Nassau [49] reports experiments to show that in infants whose weight has long kept stationary on a diet with apparently abundant protein it is possible to cause them to thrive and increase in weight by increasing their protein about fourfold. This was done by adding, for example, to a two-thirds milk mixture, 2 per cent. of a commercial albumin preparation, to an amount corresponding to 6 gm. protein per kilogram body weight, as contrasted with 1.8 gm. per kilogram in natural feeding. The findings with large groups of infants are tabulated. The failures were only in infants with acute intestinal indigestion. Indicanuria did not appear under increased protein intake as long as bowel functioning was regular.

Food Dislikes.—The influence of acquired food dislikes of childhood, and the resultant faulty nutrition, to diseases of middle life is considered by C. H. Rice.[50] He thinks that, while not capable of immediate scientific demonstration, "not a few cases of such affections of middle life as hypertension, chronic Bright's disease, visceral ptosis,

46. Shamberg and others: Summary of Research Studies in Psoriasis, J. A. M. A. **63**:729 (Aug. 29) 1914.

47. Blackfan, K. D.: Protein Hypersensitiveness in Children, Am. J. M. Sc. **160**:341, 1920.

48. Michael, J. C.: A Menace in the Low Protein Diet, Arch. Dermat. & Syph. **2**:455 (Oct.) 1920.

49. Nassau, E.: Proteins in Infant Feeding, Ztschr. f. Kinderh. **26**:270, 1920.

50. Rice, C. H.: Relation of Required Food Dislikes of Childhood to Diseases of Middle Life, J. A. M. A. **75**:100 (July 10) 1920.

gastric and duodenal ulcers, etc., are but the remote, graver manifestations of deficient diet. The commonest offense he finds in children of 4 years up living almost exclusively on carbohydrates, and with a positive dislike for milk, eggs, fresh meats or green vegetables. Such children in the South eat much bread and syrup. Talbot thinks dislikes occur in a certain group of children on a good well balanced diet. In his experience, food dislikes come in certain types of families. They come in the type of family in which there is a history of asthma, eczema and hay-fever, and sometimes urticaria. Anaphylaxis may be present, and treatment in desensitization may be desirable.

Butter-Flour Mixture.—Continental writers devote considerable attention to the use of butter-flour mixtures in infant feeding. The Czerny-Kleinschmidt mixture is given as follows: butter, 7 gm.; wheat flour, 7 gm.; sugar, 5 gm.; warm water, 100 gm. The butter is stirred over the fire until it foams and all the odor of butyric acid has disappeared. The flour is then cooked with it over an asbestos plate until the whole mass is a light brown. The warm water and sugar are then added, and the whole mixture is boiled and strained. While still warm, the mixture is added to milk in the proportions of 1 to 2 or 2 to 3. Not more than 200 c.c. per kilogram of body weight is given to the infant, and usually less.

The weight curve resembles that of the breast fed infant. It is said that neuropathic infants with the exudative diathesis and eczema thrive on the mixture, while those with the exudative diathesis and eczema alone do not thrive on it. Plantenga [51] reports favorable experiences with the mixture, and gives the histories of thirty-one babies thus fed.

Nieman and Foth [52] state that the butter-flour mixture seems to be adapted for artificial feeding of infants without severe nutritional disturbance, but for whom ordinary milk mixtures are not suitable, and that it is contraindicated where there is present acute diarrhea, or sudden great loss in weight, or acute rise in temperature from digestive disturbance.

Vitamins.—Satisfactory growth has been obtained in young rats on diets in which the almond, English walnut and filbert, respectively, furnished the essential source of protein in the ration. These nuts may also serve as the sole source of water soluble vitamin for young

51. Plantenga, B. P. B.: Butter Flour Mixture in Infant Feeding, Jahrb. f. Kinderh. **92**:375, 1920.

52. Nieman, A., and Foth, K.: The Butter-Flour Mixture, Jahrb. f. Kinderh. **93**:137, 1920.

rats, apparently affording abundant quantities. Their proteins furnish the necessary nitrogeneous complexes for the elaboration of milk in rats.[53]

According to Osborne and Mendel [54] the fresh juices of the edible parts of the orange, lemon and grapefruit contain water soluble (B) vitamin. Their potency is quite similar to that of comparable volumes of cow's milk. It is not lost by suitable modes of desiccation. A sample of grape juice was not equally efficient. Water soluble vitamin is contained also in the edible portions of apples and pears, but in relatively less quantity. Prunes appear to be richer in this factor.

Fat soluble vitamin, on the other hand, seems to be present in the juices of citrus fruits only in traces, if at all.

Daniels and Loughlin [55] report that lard and cottonseed oil contain appreciable amounts of the fat soluble growth stimulant, but it is demonstrable only when fairly large amounts are fed.

The use of orange juice and its relation to the supply of vitamin has been studied by Byfield and Daniels,[56] and by Givens and McClugage,[57] while Gerstenberger and Champion [58] report on its constipating qualities.

Hart [59] and his associates present evidence to show that the concentration of antiscorbutic vitamin in milk is dependent on the diet, summer pasture milk being richer than winter or dry feed milk. Whereas half an ounce of the former added to the basal ration protected a guinea-pig against scurvy (others required more), at least three times this amount of dry feed milk was needed. Barnes and Hume [60] compare the antiscorbutic properties of fresh, heated and dried cow's milk.

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53. Cajori F. A.: Some Nutritive Properties: of Nuts; Proteins and Content of Water Soluble Vitamin, J. Biol. Chem. **43**:583, 1920.

54. Osborne, T. B., and Mendel, L. B.: Occurrence of Water Soluble Vitamin in Some Common Fruits, J. Biol. Chem. **42**:465, 1920.

55. Daniels, A. L. and Loughlin, R.: Fat Soluble Growth Providing Substances in Lard and Cottonseed Oil, J. Biol. Chem. **42**:359, 1920.

56. Byfield, A. H. and Daniels, A. L.: The Antincuretic and Growth Stimulating Properties of Orange Juice, Am. J. Dis. Child. **19**:349 (May) 1920; **18**:546 (Dec.) 1919.

57. Givens, M. H. and McClugage, H. B.: The Antiscorbutic Property of Fruits. I. An Experimental Study of Dried Orange Juice, Am. J. Dis. Child. **18**:30 (July) 1919.

58. Gerstenberger, H. J. and Champion, W. M.: The Constipating Qualities of Orange Juice, Am. J. Dis. Child. **18**:88 (July) 1919.

59. Hart, E. B., Steenbock, H., and Ellis, N. R.: Influence of Diet on Antiscorbutic Potency of Milk, J. Biol. Chem. **42**:383, 1920.

60. Barnes, R. E., and Hume, E. M.: Comparison Between Antiscorbutic Properties of Fresh, Heated, and Dried Cow's Milk, Lancet **2**:323, 1919.

Givens and McClugage [61] report data concerning the antiscorbutic value of white potatoes subjected to various temperatures and treatment.

Canned tomato, according to Hess,[62] is the most serviceable antiscorbutic for artificially fed infants. It is well borne and comparatively inexpensive. He recommends it unreservedly in doses of 1 ounce a day. He regards canned tomatoes, from a nutritional standpoint, as a palatable solution of the three vitamins.

Mendel,[63] on feeding rats, finds daily doses of approximately 1 gm. of dried tomato serve as the source of fat soluble vitamin. As about 0.1 gm. butter fat would be otherwise necessary as an addition, the illustration indicates the relative richness of some of the edible vegetable products in fat soluble vitamin.

The antiscorbutic properties of raw beef have been studied by Dutcher [64] and coworkers. Additions of water extracts representing 5, 10, 15 and 20 gm. raw beef to a diet for guinea-pigs, of oats, water and an amount of milk sufficient to improve the diet but insufficient to prevent scurvy, did not affect the time of onset of scurvy or prolong the life of the animals. Animals receiving orange juice and beef extract presented an excellent condition.

Whipple [65] claims that the water soluble B in cabbage is not destroyed by boiling for from thirty to sixty minutes, or by boiling with acid or alkali; and that not more than one half is lost in the cooking water from cabbage and onions boiled for thirty minutes.

Miller [66] finds that the vitamins in carrots are not destroyed by heating at 115 C. for forty-five minutes. That in navy beans was reduced 40 per cent. by heating at 20 C. for thirty minutes, but the beans were somewhat overcooked. A similar loss was caused by cooking in sodium bicarbonate solution (0.5 per cent.). A large proportion of the vitamin was present in the cooking water. Muckenfuss [67] discusses the excretion of the vitamins.

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61. Givens, M. H., and McClugage, H. B.: Antiscorbutic Property of Vegetable. II. Experimental Study of Raw Dried Potatoes, J. Biol. Chem. **42**: 491, 1920.

62. Hess, A. F.: Antiscorbutic Vitamin, New York State J. M. **20**:209, 1920.

63. Mendel, L. B.: Fat Soluble Vitamin. Ibid., New York State J. M. **20**: 212, 1920.

64. Dutcher, R. A., Pierson, A. M. and Biestor, A.: Vitamin Studies. V. Antiscorbutic Properties of Raw Beef, J. Biol. Chem. **42**:301, 1920.

65. Whipple, B. K.: Water Soluble B in Cabbage and Onion, J. Biol. Chem. **44**:175, 1920.

66. Miller, E. W.: Effect of Cooking on Water Soluble Vitamin in Carrots and Navy Beans, J. Biol. Chem. **44**:159, 1920.

67. Muckenfuss, A. M.: The Excretion of the Vitamins, Arch. Pediat. **36**: 80, 1919.

Drummond [68] recommends that the somewhat cumbersome nomenclature introduced by McCollum (fat soluble A; water soluble B) be dropped, and that these substances be spoken of as vitamin A, B, C, etc. Omission of final "e" from the word makes it acceptable under the scheme of classification adopted by the American Chemical Society, which permits a neutral substance of undefined composition to bear a name ending in "in," the termination "ine" indicating that it is of a basic character.

Carotin.—Hess and Meyers have reached the conclusion that a yellow discoloration of the skin, resembling mild jaundice, except for the involvement of the sclerae, may be brought about by the ingestion of carotin. As this substance is widely distributed through a mixed diet, being a constituent of almost all our vegetable foods (carrots, spinach, oranges, egg yolks, etc.), "carotinemia" would occur most readily in those using a large amount of vegetable foods. These authors think that this pigmentation of the skin cannot be rare, and no doubt has been frequently overlooked or confused with mild grades of icterus, or attributed to some obscure metabolic disturbance. The discoloration of the skin is accompanied by a yellowing tingeing of the blood serum and plasma, and in certain cases of the urine. The pigment in the blood was identified as carotin.

Cholesterol.—In experiments on the relation of the preformed cholesterol in the food to the total cholesterol metabolism in infants, reported on by Gamble and Blackfan,[69] it was found by comparing carefully the intake and excretion of cholesterol that this substance is excreted in infants' stools without undergoing a change to coprosterol to an appreciable extent. A method for determining cholesterol in samples of milk and of stool is described.

Manganese.—Manganese was found in all tissues of the body examined by Reiman and Minot.[70] They have developed a method for the analysis of manganese in blood and tissue which is more rapid and has fewer sources of error than methods heretofore employed, and they present a series of results for the manganese content of human tissue obtained from fourteen necropsies. The liver carries the largest amount, averaging 0.170 mg. per hundred grams wet tissue.

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68. Drummond, J. C.: The Nomenclature of the So-Called Accessory Food Factors (Vitamins), Biochem. J. **14**:660, 1920.

69. Gamble, J. L. and Blackfan, K. D.: Evidence Indicating a Synthesis of Cholesterol by Infants, J. Biol. Chem. **42**:401, 1920.

70. Reiman, C. K. and Minot, A. S.: Manganese Quantitation in Biologic Material, Manganese Content of Human Blood and Tissues, J. Biol. Chem. **42**:329, 1920.

Sugars.—Miller, Bergeim, Rebfuss and Hawk [71] studied the effect of ingestion of sugars and candies on gastric secretion. They found that small amounts (10 gm. cane sugar or glucose) do not appreciably inhibit either gastric secretion or evacuation. Large amounts (3 ounces of cane sugar or glucose in concentrated solution) markedly depressed secretion and delayed the emptying of the stomach.

Candies act in proportion to their sugar content and the amounts of them ingested. This tendency is influenced by flavoring substances and added food ingredients, milk, egg, chocolate, etc., which stimulate gastric secretion. Candies should be eaten not before, but after meals. For children, hard candies which must be sucked, are preferable to cream candies because of the smaller quantity of less concentrated sugar solution derived from them. Peppermint oil used as a flavoring agent delayed stomach emptying. Chewing caramels gave rise to more voluminous gastric secretion, but delayed evacuation. Licorice gave rise to fairly abundant secretion but remained in the stomach three hours. The addition of honey to bread did not delay evacuation, but acid production was somewhat depressed.

Greenthal [72] states that the urine of all normal infants contains a determinable amount of reducing sugar. The amount is directly proportional to the amount of sugar ingested, and not dependent on the volume of urine. When the intake of sugar is constant, the excretion of sugar, both fermentable and unfermentable, is constant. As the amount of sugar in the diet is increased, the total urine sugar increases, the increase being chiefly in the fermentable portion.

Fats.—Further results in their series of studies on the fat metabolism of infants and children are reported by Holt [73] and his collaborators. In the first paper of the series (1919), it was pointed out that reliable figures on the composition of stools of breast fed infants are particularly scanty. The same investigator has seldom reported on the examination of many stools, and the majority of the results came from countries in which the living conditions and methods of feeding differ from those prevailing in this country. As many clinical conclusions regarding methods of feeding are drawn from the appearance of the stool of breast fed infants and a comparison of other stools with these, it seems most desirable to get reliable figures.

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71. Miller, R. J., Bergeim, O., Rebfuss, M. E. and Hawk, P. B.: Influence of Sugars and Candies on Gastric Secretion, Am. J. Physiol. **53**:65, 1920.

72. Greenthal, R. M.: Urine Sugar in Infants, Am. J. Dis. Child. **20**:556 (Nov.) 1920.

73. Holt, L. E., Courtney, A. M. and Fales, H. L.: A Study of the Fat Metabolism of Infants and Young Children, etc., Am. J. Dis. Child. **17**:241 (March) 1919.

Holt's first observations were made with a view to answering the following questions: (1) What is normally the percentage of total fat and what is the distribution of the fat as soap, free fatty acids and neutral fat in the stools of healthy breast fed infants? (2) What is the difference in fat content and distribution of fat between normal and abnormal stools? (3) Does the amount and distribution of fat in the stools of healthy breast fed infants vary with the per cent. of fat in the milk and with the amount of fat intake?

The material examined consisted of forty-eight collections from thirty-four different infants from 10 days to 10 months of age. Thirty of these infants were well-nourished and, with few exceptions, gaining normally. In the larger number of these cases the appearance of the stools was that typical of normal breast fed infants, largely yellow, granular or pasty, very acid to litmus, with acid, aromatic, not unpleasant odor. Others had abnormal stools—green, watery with much mucus.

In thirty-two of the cases studied the percentage of fat in the mother's milk was determined. It varied from 0.9 to 6.4 per cent. No constant relationship was found between the percentage of fat in the milk and the percentage and distribution of fat in the stool. There was, possibly, a slightly higher fat percentage in the stools of infants who were receiving milk higher in fat, but the distribution of fat in the stool did not seem to be influenced by the percentage of fat in the milk taken.

A range of fat absorption from 90.3 to 99.2 per cent. of the intake was found in healthy breast fed infants.

Notwithstanding this high absorption of ingested fat, the fat content of the stools of normal breast fed infants, according to Holt's observations, averaged 34.5 per cent. of the dried weight and frequently was as high as 50 per cent.

The soap fat in the best stools predominated over the other forms of fat, averaging 57.8 per cent. of the total fat, as determined in the dried stool. The average stool of the normal breast fed infants showed a soap fat of 43.1 per cent. of the total fat, as determined in the dried stool, which would correspond to over one-third of the total fat of the fresh stool. The neutral fat in the best stools averaged 15.9 per cent. of the total fat; in the average stool the neutral fat was 20.2 per cent. of the total fat. (The amount of neutral fat was not affected by the drying process.)

In general, Holt thought the stools were more nearly normal when the fat intake was high. Or, in other words, the normal condition in breast fed infants seemed to be a high fat intake. As the stools departed slightly from normal the fatty acids were found to be

increased at the expense of the soap fat; and in those which were markedly abnormal the neutral fat was raised at the expense of the free fatty acids.

With respect to the fat content, abnormal stools were found to be of two classes: (1) Those containing much mucus with a low fat content, and (2) diarrheal stools, in which the fat percentage is high, the result of poor absorption. Under abnormal conditions the percentage of the total fat which is in the form of soap is regularly low, while the neutral fat is high.

A second study [74] dealt with the conditions obtaining in the stools of infants fed on modifications of cow's milk. Here 128 specimens of feces were examined from seventy-two infants whose ages ranged from 2 to 18 months. It was found that in the stools of children fed on cow's milk neither the total fat nor the distribution of fat was affected by drying, a distinction which, however, might not hold in the case of acid diarrheal stools.

As the gross appearance of stools is commonly used by pediatrists as their chief guide in infant feeding, the stools were grouped in several classes according to this:

(a) Those of the type generally considered normal, formed or semiformed, smooth and homogeneous, not hard nor dry, and showing no signs of mucus. (These infants were all in good condition and gaining weight.)

(b) Those that were hard, or dry and crumbly, that is all stools that might be regarded as constipated. Most of these infants could be classed as normal healthy children; the rest though under weight were doing well at the time.

(c) Stools that were softer than normal.

(d) Stools that were not normal in appearance, being none of them smooth and homogeneous, and all showing fat curds or mucus, or both, in considerable amount, though not sufficiently loose to be classed as diarrheal. (The digestion of these children was not quite normal, and a large proportion of them subsequently developed diarrhea.)

(e) Diarrheal stools of various grades of severity. Those were arranged in two groups according to the amount of water in the daily stools, those containing more than 200 gm. being classed as severely diarrheal. Most of these children were very ill and the digestion of all was markedly abnormal.

It was found that the fat retention of infants taking cow's milk averages 88.6 per cent. of the intake, in contrast with the 95.8 per cent. retention of those taking breast milk; and that, therefore, the daily

74. Am. J. Dis. Child. **17**:423 (May) 1919.

loss of fat by infants fed on cow's milk is over twice as great as that lost by those taking breast milk, even though the actual amount of intake is somewhat less.

There was no striking relation between the fat intake and the percentage of the intake retained, except when the intake was abnormally low. The average percentage of the fat retained with normal stools was 91.3 per cent. of the intake. The retention was but little lower when the stools were somewhat harder or softer than normal, or were not homogeneous, or contained more or less mucus without being distinctly watery. As the water in the stools increased, the percentage of retention dropped markedly, reaching in severe diarrhea 58.4 per cent. of the intake.

No definite relationship was shown between the daily fat intake and the percentage of fat or the distribution of fat in the stool. The distribution of the fat showed wide variation according to the type of stool.

The average fat percentage of the dried weight in normal stools was 36.2. The hard, constipated stools showed no variation from this figure. In the stools not quite normal in appearance, the average fat percentage was slightly lower. In severe diarrhea, the fat percentage of dried weight was much higher, reaching an average of 40.7 per cent.

The soap percentage of total fat was very high in both normal and constipated stools, averaging 72.8 and 73.8 per cent., respectively. As the stools became less normal in appearance the soap fat diminished rapidly and averaged in the loose stools only 30.6 per cent. of the total fat, in the diarrheal stools 12.4 per cent. and in those of severe diarrhea only 8.8 per cent. of the total fat.

The neutral fat was less than 10 per cent. of the total fat in normal and constipated stools. It increased as the soap fat diminished and in diarrheal conditions made up about 60 per cent. of the total fat in the stool.

The free fatty acids constituted about 17 per cent. of the total fat of normal and of constipated stools. It was increased somewhat as the stools became less like the normal and in diarrheal stools was over 30 per cent. of the total fat of the stool.

A more general statement of the findings is that stools which are normal or very nearly normal in appearance show very high average values for soap, and it is only when stools show in their gross appearance evidence of digestive disturbance that the average values for soap drop below 50 per cent. of the total fat; in diarrheal stools the soap forms a very small proportion of the total fat.

The fatty acids form the next larger proportion of the total fat in good stools, the lower neutral fat indicating excellent splitting of the ingested fat. In diarrheal stools the proportion of neutral fat is greatly increased, forming, on the average, more than half the total fat.

A third study gives the findings as to fat percentage and distribution in the stools of older children receiving a mixed diet. The material examined consisted of 134 collections of feces of sixty-two children, ranging for the most part from 18 months to 5 years of age. The larger number of the children whose stools were examined were in normal condition, both as to general health and digestion. Certain abnormal children are considered separately.

The greater number of the observations reported were made on the stools of children on a diet consisting of considerable amounts of solid food and usually a pint or more of milk. In a food column accompanying the data in individual cases are enumerated those articles of the diet which contained any considerable amount of fat, such as milk, butter, eggs, cod liver oil, etc. The remainder of the diet consisted of carbohydrate foods, vegetables and fruit.

The stools were grouped according as they were constipated, normal, or not normal. The last named were, all but one, distinctly abnormal in appearance; all were acid in reaction, some extremely so; many contained considerable mucus; almost all contained undigested food and several showed evidences of fermentation. A stool was regarded as normal when it was smooth, homogeneous, not hard, showing no mucus or undigested food. The reaction of such stools to litmus was in the majority of cases decidedly alkaline; in a few it was amphotoric; in no case was it distinctly acid.

It was found that normal children on mixed diet retain on the average about 94 per cent. of the fat intake, regardless of the type of stool. The average actual retention was about 38 gm. daily. Those children taking little or no solid food, with a smaller fat intake, showed a lower actual and a somewhat lower percentage retention than did those on a general mixed diet. The highest fat loss occurred in the stools of children whose diet contained the smallest proportion of solid food and the largest proportion of milk. The average fat loss in the stools of normal children varied between 2.6 and 3.0 gm. in all the groups studied. In no group studied was there found any constant relation between the amount of fat intake and either the fat percentage of dried weight or the distribution of fat in the stools. A close agreement was found in the composition of normal and constipated stools when the diet was similar.

In the normal or constipated stools of older children whose diet consisted of milk alone or milk with bread and cereal, the fat percentage

of dried weight averaged 30.7, which is lower than the average found for similar stools of infants taking modifications of cow's milk. The soap percentage of total fat averaged 60.9, which was somewhat lower than that found in the stools of the infants. (The intake of salts, particularly those of calcium, may have some significance in determining the character of the stools, especially affecting the distribution of fat.)

The normal and the constipated stools of children on a mixed diet showed almost identical average values both for fat percentage of dried weight and for distribution of fat. The fat percentage of dried weight averaged 18.0 and 20.1 per cent., respectively, and the soap averaged 45.1 and 47.9 per cent., respectively, of the total fat—values much lower than those found when the diet contained little or no solid food.

In the acid abnormal stools of children on a mixed diet the fat averaged 15.1 per cent. of the dried weight. Both the fat percentage of dried weight and the soap percentage of total fat were much lower than in normal stools and the values for fatty acid and for neutral fat were higher.

Of the abnormal children studied, six were rachitic, and eleven were suffering from chronic intestinal indigestion, several being examples of intestinal infantilism. The rachitic children showed a slightly larger fat loss in the stools than did the normal children; their intake, however, was higher. Their actual retention, therefore, equalled or exceeded that of the normal children, and their percentage retention was only a little lower than the normal average.

In this study it is found that in the normal or constipated stools of older children whose diet consisted of milk alone or milk with bread and cereal, the fat percentage of dried weight averaged 30.7. The fat percentage of dried weight averaged 34.7 in the alkaline stools, and 24.6 in the acid stools. The values found were higher than those found for corresponding types of stools in normal children. The proportions of soap, fatty acids and neutral fat were not significantly different from those for normal children.

In the stools of children suffering from chronic intestinal indigestion the fat loss was very great, averaging 7.3 gm. daily in the alkaline stools and 8.0 gm. in the acid stools; and both the actual and the percentage retention were much lower than usual. The percentage of the intake retained averaged 79.1 when the stools were alkaline and 77.7 when they were acid. When the intake of fat was very high, the actual retention was usually as high as that found for normal children. The fat percentage of the dried weight of stools of children suffering from chronic intestinal indigestion was much

higher than for normal children, averaging 36.4 per cent. for alkaline stools and 35.3 per cent for acid stools. And the distribution of the fat was changed in that the average percentage of neutral fat was lower in both alkaline and acid stools, and the fatty acids higher, much higher when the reaction of the stools was acid.

In the case of the rachitic children, the fat percentage of dried weight averaged 34.7 in the alkaline stools, and 24.6 in the acid stools. The values thus were higher than those found for corresponding types of stools of normal children. The proportions of soap, fatty acids and neutral fat were not found to be significantly different from those for normal children. The fat intake of these children was larger than that of the normal children and their stools showed a slightly larger fat loss. Their actual retention equalled or exceeded that of the normal children, and their percentage retention was only a little lower than the normal average.

Vegetable Fats.—The digestion of some vegetable fats (nut butter and corn oil) by children on a mixed diet is the subject of a fourth study.[75] The observations would indicate that those two vegetable fats are valuable foods for children, are exceedingly well borne, and are apparently digested and absorbed with ease. The stools did not differ essentially in appearance from those of children receiving mainly milk fat, although usually somewhat softer. Except when large quantities of corn oil were taken, the fat percentages of the dried stool differed but little. The soap percentage of total fat in the stools was usually a little lower and the neutral fat a little higher with vegetable fat.

The belief seemed warranted that these articles may safely be introduced into the regular diet of children, and that to a considerable degree they may be substituted for the more expensive milk fat given as milk or butter.

As these vegetable fats are deficient in the "fat soluble A" vitamin of McCollum, which is necessary for normal growth and maintenance, they should never entirely replace milk fat in the diet. The authors also point out that the fact may not be without significance that of six children, from 80 to 95 per cent. of whose fat intake was vegetable fat, two developed styes and two others eczema on the face, which disappeared when the diet was changed to include milk fat.

Langworthy and Holmes [76] are cited as having tested the digestibility of vegetable fats in adults and having found that, with the excep-

75. The Digestion of Some Vegetable Fats by Children on a Mixed Diet, Am. J. Dis. Child. **18**:157 (Aug.) 1919.

76. U. S. Dept. Agricul. Bull. 505, 1917, etc.

tion of cocoa fat, all those tested, including cocoanut and corn oil, were digested quite as well as milk fat.

Calcium.—The calcium metabolism of infants and young children, and the relation of calcium to fat excretion in the stools was also studied.[77] As current views on these matters are often contradictory and rest most frequently on observations that are very few in number, effort was made in this study to include a sufficient number of individuals, from twenty to thirty in each series.

It was found that the total absorption of calcium oxid varied, in general, with the weight of the child; the per kilogram absorption did not vary regularly with either the age or the weight. The excretion and the absorption of calcium were in general dependent on the amount of calcium intake, from 35 to 55 per cent. of the intake being absorbed.

The average absorption of calcium oxid by healthy infants taking modifications of cow's milk was 0.09 gm. per kilogram of body weight.[78] Since the average absorption of calcium oxid by breast fed infants was about 0.06 gm. per kilogram, it may be assumed that 0.06 gm. per kilogram is the minimum normal for infants taking modifications of cow's milk.

The daily total excretion of calcium in the stools ranged from 0.34 to 1.06 gm., averaging 0.70 gm. Since the excretion and absorption of calcium were, in general, dependent on the amount of calcium intake, from 30 to 55 per cent. being absorbed, it appeared necessary, in order to insure the average absorption of 0.09 gm., that the intake of calcium oxid should be, at least, 0.19 gm. per kilogram; to insure an absorption equal to the average found for breast fed infants the intake should be at least 0.13 gm. per kilogram. When the intake of calcium oxid was very low, less than 0.10 gm. per kilogram, the absorption of calcium oxid was less than the normal calcium requirement of the body.

The best absorption was obtained when the calcium intake bore a definite relation to the fat intake, that is, when the food contained from 0.045 to 0.060 gm. calcium oxid for every gram of fat, and when at the same time the fat intake was ample, not less than 4.0 gm. per kilogram.

The relation of calcium excretion to soap excretion was not constant. Whereas, the excretion of soap was directly related to the type of stool, that is, to the water content and reaction of the stool, the excretion of calcium was closely related to the calcium intake. On the average, the normal and the constipated stools, with high soap content, were found when the intake of calcium was high, and, therefore they showed the higher excretion of calcium. However, constipated stools which contained more soap than normal stools, had lower calcium content. Nonhomogeneous stools, with the lowest average soap content, showed the same content of calcium not held as soap as did the constipated stools, with the highest soap content. The calcium that could be lost as soap was never a large proportion of the calcium intake. Even in the stools containing the most soap it was found to be less than three-tenths the calcium intake. The calcium lost as phosphate was shown not to be increased in soapy stools. The calcium percentage of the total solids varied, as a rule, with the water content of the stools, diminishing as the water increased.

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77. Holt, L. E., Courtney, A. M. and Fales, H. L.: Calcium Metabolism of Infants and Young Children, and the Relation of Calcium to Fat Excretion in the Stools, Am. J. Dis. Child. **19**:97 (Feb.) 1920.

78. Range from 0.013 to 0.133 gm.

An excessive calcium intake apparently did not increase the calcium absorption, the excess being excreted. The calcium absorption was much lower when diarrhea was present. The absorption of calcium was regularly increased by the administration of cod liver oil, unless diarrhea was present.

The calcium absorption by rachitic infants was much lower than the absorption by healthy infants, the average for the group being 0.042 gm. per kilogram, less than one half that for normal infants. The few observations in infants recovering from rickets showed a calcium absorption that was higher than the normal average. These infants had received cod liver oil for a considerable period.

A second paper on calcium metabolism and the relation of calcium to the fat excretion deals with the conditions obtaining in the case of children on a mixed diet.[79]

In such children the intake of calcium oxid averaged 0.108 gm. per kilo, ranging from 0.043 to 0.178 gm., in about half the cases being not more than 0.10 per kilo. The intake thus was lower than in the case of infants; the absorption also was lower. With children on a mixed diet, the absorption of calcium per kilo averaged, when the intake was adequate, 0.055 gm. calcium oxid per kilo. The average daily excretion of calcium oxide was 0.87 gm., being dependent more on the amount of the calcium intake than on the type of stool.

The percentage of calcium intake absorbed when the intake exceeded 0.09 gm. per kilo averaged 40.4; when the intake was 0.09 gm. or less, the absorption averaged only 20.3 per cent. Therefore, it may be inferred that an intake of at least 0.09 gm. calcium oxid per kilo is necessary to insure a good absorption by children taking a mixed diet.

The best absorption occurred when the intake of fat exceeded 3.0 gm. per kilo, and when at the same time there was in the diet from 0.03 gm. to 0.05 gm. calcium oxid for every gram of fat.[80] When the intake was only 0.09 gm. per kilo, or less, the absorption rarely exceeded 0.03 gm. per kilo, and in several instances there was a negative balance; the average being only 0.015 gm.

When calcium in the form of chalk mixture (calcium carbonate) was added to the diet, there was a greatly increased absorption of calcium. The absorption was not increased when calcium acetate or calcium phosphate was added.

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79. Holt, L. E., et al: Children Taking a Mixed Diet, Am. J. Dis. Child. **19**:201 (March) 1920.

80. The statement is also made that the excretion of calcium "was not at all related to the fat intake."

The two factors chiefly affecting the percentage of calcium in the stools were the amount of calcium intake and the reaction of the stools. The percentage of the total solids was lower in acid than in normal or constipated stool. The calcium lost as soap was in most cases an insignificant part of the calcium intake.

The absorption of calcium by children with chronic intestinal indigestion was extremely low; as it was also in children with active rickets.

Cod liver oil increased the absorption of calcium, except in cases in which the intake of calcium or of fat was very low. The substitution of vegetable fats for milk fat did not affect the calcium metabolism of children taking a mixed diet.

According to Grimm [81] in normal children at the end of the first and at the beginning of the second year, the amount of feces varies from 5.5 to 8.5 gm. for each 100 gm. food taken; from 6.0 to 9.6 gm. for each kilogram of body weight.

The behavior of calcium in the body and its distribution under various conditions of health and disease, and under special feeding continues to attract much attention. Sherman [82] has attempted to determine the calcium requirement of adult men.

Just as the protein need has been shown to average 45 gm. a day for a person of 70 kg. weight, and the phosphorus need is estimated at 0.9 gm. a day, so the new data for calcium requirement give a mean result of 0.45 gm. Expressed differently, this would mean that in order to furnish those essential nutrients in relative proportions corresponding to the needs of the adult body, a diet should contain at least 1 gm. calcium for every 100 gm. protein.

A study of the food supplies of American families and larger groups has shown that this ratio does not obtain therein. However there was probably no deficiency of calcium in the usual diet, since the percentage of protein was unnecessarily large. When calculated to the usual basis of nutrients for one man per day, the average protein content in 224 presumably typical American dietary studies, was found to be 106 gm. and the average calcium content 0.74 gm.

A method is described by Kramer and Howland [83] by which the calcium in 2 c.c. of serum may be determined with a maximum error of 3 per cent. and often less than 1 per cent. By the use of this

81. Grimm, G.: Abnormally Large Amounts of Feces in Young Children, Monatschr. f. Kinderh. **18**:193, 1920.

82. Sherman, H. C.: Calcium Requirement of Maintenance in Man, J. Biol. Chem. **44**:21, 1920.

83. Kramer, B., and Howland, J.: Method for Determinations of Calcium in Small Quantities of Blood Serum, J. Biol. Chem. **43**:35, 1920.

method they are able to show that the concentration of calcium in the serum of normal adults is strikingly constant, and amounts to 9 or 10 mg. per hundred cubic centimeters.

Handovsky[84] reports that in twelve adults who manifested the Chvostek phenomon and in nine who were free from it, the calcium content of the blood lay within the same range in all; thus contradicting, in adults at least, Sherman's statement that the calcium content is low when this sign is positive.

In a rather popular article Peckham[85] states that calcium is an essential part of the human economy, constituting approximately 50 per cent. of the inorganic matter of bone, and being an important part of the structures in ligaments, muscles and the nervous system; and he adds that when calcium metabolism is interfered with there may result a deficiency, in which case, the bones, muscles, nerves and ligaments are deprived of their calcium. "Under these conditions, there may result scoliosis, knock knees, bowlegs, flat feet, etc." Little satisfactory evidence for these statements is given, and in the discussion of the paper many diverse opinions were expressed.

The frequent use of carrots as an addition to broths in the feeding particularly of rachitic infants, lends additional interest to the studies recently made by Rose[86] on healthy young adult women, to determine the utilization of the calcium of carrots by the human body. In three out of four cases, she found that there was a positive calcium balance in the carrot diet, when the calcium intake was close to the estimated minimum for equilibrium; in the fourth case the loss was small. When approximately 55 per cent. of the calcium was derived from carrots, one subject had practically the same retention as on a diet on which 70 per cent. of the calcium was derived from milk. It seems possible, therefore, to meet the requirements of the adult human organism largely, if not wholly, from carrots.

Rickets and Tetany.—Nathan,[87] inquiring whether or not rachitis is a deficiency disease, summarizes some recent research which seems to answer the question affirmatively.

The diagnostic significance of craniotabes and beading of the ribs as signs of rickets is considered in a paper by Schwarz.[88]

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84. Handovsky, I.: Calcium Content of Blood, Jahrb. f. Kinderh. **91**:432, 1920.

85. Peckham, F. E.: Many Orthopedic Deformities Due to Calcium Deficiency. A Direct Result of Sterilized and Pasteurized Food, J. A. M. A. **75**: 1317 (Nov. 13) 1920.

86. Rose, M. S., et al: Experiments on Utilization of Calcium of Carrots by Man, J. Biol. Chem. **41**:349, 1920.

87. Nathan, M.: Is Rachitis a Deficiency Disease, Press méd. **28**:577, 1920.

88. Schwarz, H.: Craniotabes and Beading of the Ribs as Signs of Rachitis, Am. J. Dis. Child. **19**:384 (April) 1920.

Howland and Park [89] show that a definite correlation exists in rickets between the roentgen-ray findings and the actual pathologic conditions. As calcium deposits in cartilage cast well defined shadows, the effectiveness of cod liver oil as a therapeutic agent in rickets could be demonstrated. In animal experiments a beginning calcium deposit was demonstrated two days after beginning the administration of cod liver oil; in human beings the calcium deposit in the cartilage was definitely demonstrable at the end of three weeks.

In the discussion, Marriott stated that he had prepared a solution containing all the inorganic constituents of blood plasma, an "artificial blood," containing phosphates, lime, magnesium salts, sodium bicarbonate and carbon dioxid, the latter being at a tension of 40 millimeters. This solution was perfectly clear, but a precipitate occurred when a portion of the carbon dioxid was removed, or if more bicarbonate, calcium salts, or inorganic phosphate were added. Only by increasing the amount of phosphate in the solution was a precipitate obtained that had a composition the same as that of bone. A very small increase in the amount of phosphate in solution caused a very considerable precipitate of the substance having the approximate composition of bone. A very small increase in the amount of phosphate in solution caused a very considerable precipitate of the substance having the approximate composition of bone. Marriott added that it would seem likely, therefore, that the method by which bone is laid down is by an increase in the amount of phosphate present at some points. Howland added that after feeding cod liver oil the phosphorus content of the blood was found greatly increased.

Jacobowitz [90] found the calcium content of the blood in children with tetany decidedly lower than in twenty-one children free from signs of tetany. Even when the therapeutic results seemed excellent, no influence on the calcium level in the blood could be detected on administration of calcium by mouth, in the tetany cases and others.

Hoobler [91] reports that, in general, the retention of calcium, magnesium and phosphorus was found to be considerably diminished in children subject to convulsive disorders when compared with normal controls. He thinks that in such children an attempt should be made to discover the mineral deficiency, and if such a deficiency is found to bring the condition up to normal.

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89. Howland, J. and Park, E. A.: Proc. Am. Ped. Soc., 1920.

90. Jacobowitz, S.: Calcium Content of Blood in Tetany, Jahrb. f. Kinderh. **92**:256, 1920.

91. Hoobler, B. R.: Proc. Am. Pediat. Soc., 1920.

Some years ago Howland and Marriott [92] showed that in infants suffering from tetany, the amount of calcium in the blood serum might be decreased 50 per cent.

Clark [93] reports that feeding a calcium-rich diet to animals had no effect on the calcium content of their blood. Intravenous or subcutaneous injections of lime salts brought about a transitory increase. Considerable evidence might be cited to show that feeding of calcium rich or calcium poor diets does not make any appreciable change in the amount of this element found in the blood, bones or other tissues.

Brown, MacLachlan and Simpson [94] report that administration of cod liver oil and phosphorus produced an increase in blood calcium in case of tetany, with a corresponding reduction in the mechanical and electrical signs, within a period of from ten to seventeen days.

Constitutional reactions were produced in nine patients following intravenous injection of calcium lactate in 1.25 gm. doses. It varied from slight drowsiness to almost complete collapse, accompanied by dyspnea. These signs disappeared usually between one and seven hours, severe reaction being recovered from more slowly. A temporary absence of both the electrical and mechanical signs of tetany was noted, usually lasting from seven to ten hours.

Unless supplemented by the administration of cod liver oil and phosphorus, apparently no beneficial therapeutic effect was exerted. Combination of the two methods resulted in a little more rapid reduction of the tetanoid symptoms than did the use of cod liver oil and phosphorus alone.

Grosser [95] reports the details of a study on the effects of injecting different salts of calcium and phosphorus parenterally in ten children on comparable diets in an effort to see which might be most readily assimilated by a child with a tendency to rachitis. He was not able to draw any general conclusions.

Schiff and Peiper [96] report that injection of pilocarpin in four infants from 2 to 4½ months old induced profuse sweating and retention of a larger proportion of calcium (one excepting), and epinephrin

92. Howland, J., and Marriott, W. McK.: Quart. J. M. **11**:296, 1917.

93. Clark, G. W.: Effect of Hypodermic and Oral Administration of Calcium Salts on the Calcium Content of Rabbit Blood, J. Biol. Chem. **43**:89, 1920.

94. Brown, A., McLachlan, J. F. and Simpson, R.: Effect of Intravenous Injections of Calcium in Tetany and Influence of Cod Liver Oil and Phosphorus on Retention of Calcium in Blood, Am. J. Dis. Child. **19**:413 (June) 1920.

95. Grosser, P.: The Metabolism in Rachitis, Ztschr. f. Kinderh. **33**:141, 1920.

96. Schiff, E. and Peiper, A.: Calcium Metabolism in Infants, Jahrb. f. Kinderh. **93**:160, 1920.

reduced the retention of calcium (one exception). They think this demonstrates a new and close connection between the calcium metabolism and the nervous system.

Sharpe,[97] continuing the earlier studies of Noel Paton and Findlay[98] studied the guanidine content of the feces of normal children and those suffering from tetany and found quantities of this base averaging 0.007 per cent. of the moist feces in normal children and 0.075 per cent. in cases of tetany. The actual amount excreted daily in the latter cases averaged 0.018 gm. as dimethylguanidine.

Benedict[99] and also Talbot[100] discuss the caloric requirement by children from birth to puberty.

The Circulation.—Recent investigations indicate that the capillaries play a more active part in the volume distribution of the blood than has been supposed. The fact that they have not muscular coats like arterioles and veins, need not necessarily indicate that they play an entirely passive rôle and become dilated or collapsed according to the different masses of blood that reach them. It is possible that the cells forming their delicate walls are contractile, this attribute being common to living cells and but highly specialized in muscle cells.

Krogh[101] has summarized considerable evidence tending to show that these capillaries contain contractile elements of some sort within themselves and that they function especially in the direction of active dilatation.

The behavior of capillaries toward certain drugs leads to the conclusion that dilator effects, for example, can be produced independently of any extrinsic nerve supply or alterations in the arterial circulation. In studies on the capillaries of the tongue of the frog, it appeared that only to a slight extent, if at all, can capillary tonus be of a nervous nature, since it is generally maintained after section and degeneration of the nerves that might be involved. The maintenance of this tonus is, however, dependent on the blood supply. When a vessel gets no blood, its tone diminishes, and finally it relaxes, thus admitting blood and allowing the tone to be restored. Krogh concludes that every capillary must alternately open and close, thus providing uniform irrigation for the whole tissue when a sufficient period of time is con-

97. Sharpe, J. S.: The Guanidine Content of Feces in Idiopathic Tetany, Biochem. J. **14**:46, 1920.

98. Quart J. Exper. Physiol. **10**:3 and 4, Pt. 4, 1917.

99. Benedict, F. G.: Energy Requirements of Children from Birth to Puberty, Boston M. & S. J. **181**:107, 1919.

100. Talbot, F. B.: The Caloric Requirements of Normal Infants and Children from Birth to Puberty, Am. J. Dis. Child. **18**:229, 1919.

101. Krogh, A.: Studies in Capillariomotor Mechanism, etc., Am. J. Physiol. **53**:399, 1920.

sidered. The substance responsible for the tonic action of the blood is not oxygen.

The possibility of a chemical regulation of the blood vessels and blood flow is thus emphasized. Metabolites, or drugs, which produce a vasomotor action not clearly attributable to an effect on well known vasomotor nerves may act on the less well known local nervous mechanism, or perhaps, even directly on the musculature, or on the cells of the walls themselves, in finer vessels.

Hooker [102] would conceive of a primitive chemical mechanism of control, with superimposed on this a highly organized nervous mechanism adapted to quick and efficient response. On the surface of the body, where rapid responses to changes in environment may be more necessary, the latter mechanism of control may be more highly developed. In the glands and other deeper body tissues, generally, where reaction is conceivably of less significance, the primitive chemical control may be relatively more important.

This extension of our conception of vasomotor control to all parts of the vascular system greatly complicates our ideas of the part played by the vascular system in normal physiology and in disease. The introduction of chemical factors that act locally further complicates the matter. Nevertheless, it is a conception toward which various pathologic studies have tended.

Utheim [103] has studied the blood and its circulation in well and sick infants. He finds that the serum protein in normal infants is from 6 to 6.5 per cent. and remains at this level until the tenth or eleventh month, after which it begins to rise, and by the fifteenth month has reached the level found in adults, 8 per cent. Premature infants show a low protein percentage in the blood. Infants suffering from diarrhea and vomiting have a high percentage of protein, the result of blood concentration. Otherwise, infants suffering from various diseases show, with the exception of exudative diathesis and nephritis, no noteworthy change in the blood protein concentration.

Athreptic infants show a low protein percentage in the blood. This in some instances seems due to lack of power on the part of the organism to build up protein, in other instances to overfeeding with carbohydrates. Athreptic infants also show a very low rate of blood flow which is thought to be due, in some instances, partly to the diminished blood volume, in other instances to constriction of peripheral

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102. Hooker, D. R.: The Functional Activity of the Capillaries and Venules, Am. J. Physiol. **54**:30, 1920.

103. Utheim, K.: Study of Blood and Its Circulation in Normal Infants, etc., Am. J. Dis. Child. **20**:366 (Nov.) 1920.

small vessels in order to accomplish the distribution of the blood to the internal organs. This low blood flow is not usually accompanied by a lowering of blood pressure.

It is suggested that the high water content of the organism in both premature and athreptic infants is an important factor in the low immunity they show, predisposing these infants to multiple infections.

Acidosis and Alkalosis.—The prevailing conception of asphyxial acidosis is, according to Haggard and Henderson,[104] in many respects diametrically opposed to the facts. Under low oxygen overbreathing occurs before the blood alkali is appreciably reduced. It is improbable that such a condition as "lactic acid acidosis" ever occurs in life. Intravenous injections of lactic acid do not induce an acidosis at all commensurable with the amount administered. An increase of lactates in the blood or urine is probably an indication, not of acidosis, but of low ratio of carbonic acid sodium bicarbonate, that is, alkalosis.

The conditions attending the production of tetany through forced respiration are reported by Grant and Goldman.[105] Forced respiration causes symptoms of tetany to appear in the human subject; carpopedal spasm, Chvostek's sign, Trousseau's sign, Erb's sign, and (in one instance as observed by them) tetanic convulsion. As a result of the fall of alveolar carbon dioxid tension produced by overventilation, there is a reduction of the carbon dioxid capacity of plasma, a change in the reaction of the urine to the alkaline side, a decreased excretion of ammonia, and a slight increase in the calcium content of the serum. These authors believe that the underlying factor in the production of tetany of forced respiration is the alkalosis.

Seham [106] writes on the acidotic state of normal new-born infants, with special reference to carbon dioxid tension, alkali tolerance and acetonuria.

A paper by Gamble and Goldschmidt [107] deals with the relation of creatinuria to acidosis.

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104. Haggard, H. W. and Henderson, Y.: Hematorespiratory Functions. III. The Fallacy of Asphyxial Acidosis, J. Biol. Chem. **43**:3, 1920.

105. Grant, S. B. and Goldman, A.: Study of Forced Respiration; Experimental Production of Tetany, Am. J. Physiol. **52**:209, 1920.

106. Seham, M.: The Acidotic State of Normal Newborns with Special Reference to the Alveolar Carbon Dioxid Tension, Alkali Tolerance and Acetonuria, Am. J. Dis. Child. **18**:42 (July) 1919.

107. Gamble, J. L. and Goldschmidt, S.: Creatinuria in Infants. I. Relation of Creatinuria to Acidosis. Elimination of Ingested Creatin and Creatinin, J. Biol. Chem. **40**:199, 1919. Relation of Protein Intake to Urinary Creatin, **40**:215, 1919.

PATHOLOGY

The popular opinion that during and after a war a great many more boys are born than girls has recently been tested and found to be incorrect.

During the first decade of the twentieth century the excess of male over female births ranged in Germany and Austro-Hungary and other countries from 5 to 6 per cent. In England, the apparent excess was only 3.9 per cent. for the years 1906-1910, but this is shown to be due to official conditions of notification. To offset this excess of male births, there is a higher mortality of male new-born than that of female new-born. According to the German vital statistics for the same decade, 202 boys died, as compared with 170 girls, out of every thousand. This gave a male excess mortality of thirty-two to each 1,000 living new-born. According to the 1912 statistics, the greatest excess of mortality occurs during the first month, and, calculating the excess mortality of boys in percentage of girl mortality, according to the principal causes of death, the following figures for the male excess mortality are obtained; defective vitality, 23.8 per cent.; diseases of the digestive organs, 20.8 per cent.; diseases of the respiratory organs (exclusive of pneumonia) 18 per cent.; pneumonia, 17 per cent. and infectious diseases 0.0 per cent. Professor Silbergleit, of the Berlin office of statistics, concludes, therefore, that defective vitality and diseases of the digestive organs are the main causes of the excess male mortality, and that better care and nursing, appropriate feeding and careful supervision can help to combat the effects of the apparently weaker constitution of male infants. Behla, former director of the Prussian office of statistics, reports no extraordinary excess in birth of boys up to 1917, and the usual proportion maintained during 1915. Schanta, of Vienna, reported a proportion of 100-102 for 1915-16; Bumm, of Berlin, 100-116; Kronig of Freiburg found no increase of boys.[108]

Schwarz [109] presents a paper on infant and child mortality, including miscarriages and still-births.

Aschenheim [110] describes what is supposed to be the first authentic instance of roentgen-ray injury to a human fetus. The case is that of an imbecile boy with an unusually small head, sunken and nearly blind eyes, and occasional convulsions. The otherwise healthy mother, at 37, was given deep roentgen-ray treatment for myoma of the

108. J. A. M. A. **75**:888 (Sept. 25) 1920.

109. Schwarz, H.: Infant and Child Mortality, Including Miscarriages and Still Births, Am. J. Dis. Child. **19**:249 (March) 1920.

110. Aschenheim, E.: Roentgen-Ray Injury of Human Fetus. Arch. f. Kindrh. **68**:131, 1920.

uterus, and conception must have occurred about a month before the treatment was begun.

Baldwin [111] describes the anomalies produced by exposure of the protoplasm of frog's eggs to roentgen rays.

Cerebral Hemorrhage.—It has been known that cerebral hemorrhage in the new-born may be a cause of infantile death and spastic paraplegia in those who survive.[112] It has also been suspected that symptomless and probably lesser degrees of meningeal bleeding are quite common. Recently, however, Rodda [113] reports from statistics of the New-Born Clinic of the University of Minnesota that postmortem examinations reveal cerebral hemorrhage in more than 50 per cent. of all infants that die intrapartum or during the first few days of life. It was notable that these findings were often made following noninstrumental or even easy deliveries. They were especially frequent following breech presentations and in premature births. The factor of asphyxia neonatorum was not always present. It was further noted that at necropsy the blood was often found only slightly coagulated or even fluid.

Seitz's studies on frozen sections of the skull are cited in support of a view that the massive hemorrhages in the still-born or in infants that die in the first hours of life result from the rupture of large veins, sinuses or tears of the tentorium. There remain, however, the majority of cases of cerebral hemorrhage developing insidiously and not until several days after birth, causing symptoms of increasing intracranial pressure, convulsions and death. In these cases, necropsy revealed a large clot with a thick center, thin edges, usually unilateral, and covering the parietal area; and there was no demonstrable source of hemorrhage. Further examination revealed multiple hemorrhages throughout the body in locations where it was hard to conceive trauma from normal birth forces or obstetric procedures.

Rodda [114] finds the explanation of these cases in a delayed coagulation time for the blood, hemorrhagic disease. With his method he finds that the average coagulation time in the normal new-born is seven minutes, with a normal range of from five to nine minutes; the aver-

111. Baldwin, W. M.: Artificial Production of Monsters Demonstrating Localized Defects as Result of Injury from Roentgen Rays, Am. J. Physiol. **52**:296, 1920.

112. McNutt, L. J.: Double Infantile Spastic Hemiplegia, with the Report of a Case, Am. J. M. Sc. **89**:58, 1886. Intracranial Hemorrhage in Children, New York M. J. **41**:104, 1885.

113. Rodda, F. C.: The Coagulation Time of Blood in the New-Born, with Especial Reference to Cerebral Hemorrhage, J. A. M. A. **25**:452 (Aug. 15) 1920.

114. Rodda, F. C.: Studies with a New Method for Determining the Coagulation time of the Blood in the New-Born, Am. J. Dis. Child. **19**:269 (March) 1920.

age bleeding time, with Duke's method, is from three to three and one-half minutes, with a normal range of from two to five minutes. There is a prolongation of coagulation and bleeding times from the first to the maximum on the fifth day of life, with a return to the average first day determination before the tenth day.

It is significant that this coincides with the age incidence of hemorrhagic disease and cerebral hemorrhage, and evidences of hemorrhage appeared when a prolonged bleeding time accompanied a delayed coagulation time. Therefore, Rodda concludes that a more frequent cause of cerebral hemorrhage is mild trauma plus hemorrhagic disease of the new-born, accompanied by findings of delayed coagulation time and prolonged bleeding time.

The latter evils can be controlled by the subcutaneous injection of whole blood. In severe cases surgery should be employed early. Routine blood examination of the new-born, or at least of those presenting unusual symptoms, is advocated. (This is advocated also by Foote.[115])

Sinus Thrombosis.—Hamburger[116] analyses twenty-six cases of sinus thrombosis in young children. All were under 4 months of age, the youngest being 11 days old. Sinus thrombosis usually developed so insidiously during the course of a general infection as to be a surprise at necropsy. Symptomatic convulsions and signs of encephalitis reveal the bacterial invasion of the brain. In a few cases, there was stasis in the peripheral veins, with edema in the face and head. Somnolency and cerebral vomiting were not characteristic.

Miscellaneous Lesions.—An instance of aneurysm of the ascending arch of the aorta in a boy, aged 13 years, reported by Heiman.[117] The study is a clinical one, but is illustrated by photographs tracings and roentgen-ray findings.

Holt[118] reports the occurrence of primary sarcoma of the thymus in a child aged 6 months. The case was regarded as one of severe secondary anemia, with symptoms dating back only four weeks, and consisting of palor and slight fever. The child presented none of the symptoms attributed to enlargement of the thymus. At necropsy, a sarcomatous thymus weighing 36 gm. was found, also lesions of a similar nature in a lymph node, in the spleen and in the lungs.

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115. Foote, J. A.: Hemorrhagic Tendency as Frequent Cause of Cranial hemorrhage of New-Born, Am. J. Dis. Child. **20**:18 (July) 1920.

116. Hamburger, R.: Vascular Thrombosis in Young Children. Jahrb. f. Kinderh. **91**:439, 1920.

117. Heiman, H.: Aneurysm of the Ascending Arch of the Aorta in a Boy of Thirteen. Arch. Pediat. **36**:543, 1913.

118. Holt, L. E.: Primary Sarcoma of Thymus. Proc. Am. Pediat. Soc. 1920.

The general subject of abscess of the lungs in infants and children has been studied by Wersler and Schwarz,[119] and the presence of foreign bodies in the lungs by Graham.[120]

An instance of liver abscess in an infant 1½ years old is reported by Carvallo.[121] The child had symptoms of chronic dysentery and an abscess in the liver and right lower lobe. This was evacuated after resection of two costal cartilages, and the child recovered under emetin treatment.

A case of hepatoma in an infant is reported by Wollstein and Mixall.[122]

Tull and Alam [123] report the occurrence of a lipoma in the breast of a girl, aged 11 years. It weight 51 pounds.

An instance of carcinoma of the ovary in a girl 11 years old is reported by Ridout.[124]

Septicemias.—Dwyer [125] reports a case of gonococcemia in a female child, aged 23 months, with malignant endocarditis and a metastatic abscess in the sacral region. The causative organisms were recovered from the blood, and also, apparently, from the vagina and the abscess. The cardiac lesions were confined to the mitral valve. On the free border of each cusp there were greenish-yellow cauliflower vegetations. The posterior cusp was perforated, the circular opening measuring 6 mm. in diameter. The edges of the perforation were bordered with the vegetation.

The case is reported because of the age of the patient, ulcerative endocarditis being uncommon under 5 years, and because of the superficial abscess in the lumbar region of gonococcic origin. Dwyer refers to the series of cases of endocarditis in children assembled by Gilbert (197 cases), Satterwaite (100 cases) and Dunn (304 cases), and states that streptococcus ulcerative endocarditis has been reported in an infant aged 6 months, that Huber has recovered *Streptococcus attenuans* in an infant 1 year old with endocarditis, and that *Streptococcus viridans* is the common offender in "rheumatic" endocarditis and in

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119. Wersler, H. and Schwarz, H.: Abscess of the Lungs in Infants and Children, Am. J. Dis. Child. **19**:137 (Feb.) 1920.

120. Graham, E. E.: Foreign Bodies in the Air and Food Passages. Am. J. Dis. Child. **19**:119 (Feb.) 1920.

121. Carvallo, C. J.: Dysenteric Abscess in Infant, Crón. méd., Lima **37**:208, 1920.

122. Wollstein, M. and Mixall, H. R.: Report of Case of Hepatoma in an Infant, Arch. Pediat. **36**:268, 1919.

123. Tull, J. C. and Alam, M.: Large Lipoma in Child. Indian M. Gaz. **55**:299, 1920.

124. Ridout, E. A. S.: Primary Carcinoma of Ovary at Age of 11. Brit. M. J. **2**:380, 1919.

125. Dwyer, H. L.: Malignant Endocardites and Metastatic Abscess in Gonococcemia, etc., J. A. M. A. **75**:1643 (Dec. 11) 1920.

those cases termed slow endocarditis or endocarditis lenta. In the three series only one case was found to be due to the gonococcus.

An instance of *Staphylococcus aureus* septicemia and endocarditis in a boy 11 years old is reported from the Children's Department of the University of California Medical School.[126]

Other than for an attack of moderately severe tonsillitis eight months earlier, his history was essentially negative, except that four weeks before admission he had fallen, and injured his right knee over the patella. An abscess had formed and two weeks later been incised and drained, apparently healing well. Coincidently, however, there developed malaise and listlessness. Four days before admission, fever and precordial pain were first noted, and two days later dyspnea became pronounced. A rough systolic murmur was present, and a pericardial friction rub. The roentgen ray showed a pericardial effusion. Cultures from the nose, throat and blood yielded *Staphylococcus aureus,* as did the cultures from serofibrinous fluid withdrawn in pericardial puncture. Decompensation soon set in and death followed.

Necropsy showed a heart weighing 240 gm., and covered with a tough grayish-yellow membrane. On section, the myocardium was dark red in color but showed no macroscopic evidence of general myocarditis. The valves were normal. One centimeter beneath the right cusp of the tricuspid valve was a small slightly elevated yellowish area extending from the endocardium. This was found to be the apex of a funnel-shaped abscess extending in the direction of the aortic semilunar valve. The aorta presented a number of yellow atheromatous patches in the ascending and transverse portions of the arch. There were pyemic abscesses in the kidneys.

Brennemann[127] discusses the evidence and significance of rheumatic nodules in children.

Meuron[128] reports a case of erythema nodosum and acute endocarditis in a boy, aged 8 years.

Scurvy.—Infantile scurvy is the subject of two papers by Comby.[129]

Scorbutic beading of the ribs is discussed by Hess and Unger.[130] An instance of cerebral hemorrhage in scurvy is reported by Sammis,[131] and an instance of scorbutic hematuria by Eaton.[132]

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126. Case Histories from the Children's Department University of California Medical School and Hospitals. California State J. M. 1920.

127. Brennemann, J.: The Incidence and Significance of the Rheumatic Nodules in Children. Am. J. Dis. Child. **18**:179 (Sept.) 1919.

128. Meuron, D. Erythema Nodosum and Acute Endocarditis in a Boy of Eight. Arch. de méd. d. enf. **22**:428, 1919.

129. Comby, J.: Infantile Scurvy. Arch. de méd. d. enf. **22**:225, 281, 1919.

130. Hess, Alfred F. and Unger, Lester J.: Scrobutic Beading of the Ribs; Am. J. Dis. Child. **19**:331 (May) 1920.

131. Sammis, J. F.: A Case of Scurvy with Cerebal Hemorrhage, Arch. Pediat. **36**:274, 1919.

132. Eaton, P. J.: Arch. Pediat. **36**:358, 1919.

Megacolon.—A paper by A. E. Meyers [133] deals with dilatation of the colon in children, and Kerley [134] contributes a paper on roentgen-ray demonstration of abnormalities of the gastro-intestinal tract in children. Casati's [135] experience would indicate that megacolon may be more common than is generally realized, but escapes detection unless it becomes obstructed. He reports cases (one in a child) in which the weight of the feces had dragged the colon down farther and farther until the sigmoid loop had been kinked and total obstruction resulted. The obstruction was overcome at once by raising the foot of the bed quite high so that the pelvis sloped sharply down toward the shoulders, the weight then dragged down the other way, unkinking the loop. Almost immediately, the contents of the megacolon were expelled with great force, thus confirming the correctness of the explanation. The rectum earlier had been found empty, but through its wall could be palpated the mass of the distended colon.

Magliani [136] describes Francioni's method of treating congenital megacolon in children by introducing a long flexible tube into the rectum. This overcomes the kink, which is assumed to be due to the sigmoid loop being unusually long or for some reason getting kinked or otherwise occluded. By allowing for the escape of gases and feces, the immediate disturbance is corrected and opportunity given for the anomaly to be outgrown. Magliani leaves the tube in place for a number of hours (up to thirty-six hours) and reintroduces it at intervals of four or five days, or oftener, as the symptoms develop.

In two cases which are reported in detail—an infant of 19 days, and a child of nearly 3 years—the tube seemed to meet an obstacle past which it had to be worked. A few days treatment sufficed for the infant, but in the older child treatment had to be kept up intermittently for several months.

Porter, Morris and Meyer [137] discuss certain nutritional disorders of children which are found associated with a putrefactive intestinal flora.

Intestinal Parasites.—DeBuys and Dwyer [138] have studied the per-

133. Meyers, A. E.: Dilatation of the Colon in Children; Am. J. Dis. Child. **19**:167 (March) 1920.

134. Kerley, C. G.: Roentgen-Ray Demonstration of Abnormalities of the Gastro-Intestinal Tract in Children, Am. J. Dis. Child. **19**:277 (April) 1920.

135. Casati, E.: Item from Megacolon, Policlinico **27**:699, 1920.

136. Magliani, I.: Treatment of Congenital Megacolon, Riv. d. clin. Pediat. **18**:321, 1920.

137. Porter, L.; Morris, G. B. and Meyer, K. F.: Certain Nutritional Disorders of Children Associated with a putrefactive Flora, Am. J. Dis. Child. **18**:254 (Oct.) 1919.

138. DeBuys, L. R., and Dwyer, H. L.: Study of the Stools in Children's Institutions Showing the Incidence of Intestinal Parasitic Infection, Am. J. Dis. Child. **18**:269 (Oct.) 1919.

centages of intestinal parasitic infection in children of Southern institutions.

An instance of migration of a round worm into the ear is reported by Coussieu.[139]

Ascaris as a cause of pulmonary disease was discussed by Ransom [140] last year.

Syphilis.—The subject of prenatal syphilis is considered by Kolmer.[141] He cites Vedder's estimate that from 10 to 28 per cent. of men from the class of unskilled labor and the trades, varying in age from 18 to 40 years, are syphilitic, as well as 10 per cent. of men of better education. He also states that among presumably healthy young women the percentage of syphilitic infections fluctuates between 2 and 20 per cent., depending on age, marital condition, education and social status, the percentage of infection increasing as one descends in the social scale. The incidence in negroes is estimated to be at least double the figure for whites.

Probably one miscarriage out of every ten involves a syphilitic individual. According to Jeans,[142] at least 75 per cent. of the offspring of syphilitic families are infected. Thirty per cent. of the pregnancies terminate in death at or before term. Moreover, among syphilitic children the death rate is given as about double the normal, i. e., 30 per cent. It is estimated that only about 17 per cent. of all pregnancies in syphilitic families result in living nonsyphilitic children that survive the period of infancy.

It is found that from 2 to 6 per cent. of hospital and dispensary children give a positive Wassermann reaction, with higher percentages among backward, mentally deficient and sick children. Kolmer says it would seem safe to assume 5 per cent. of syphilis in the infant population, so far as detectable.

Cerebral involvement in hereditary syphilis was considered by Jeans a year ago.

Tuberculosis.—Austin [143] discusses the occurrence of tubercle bacilli in the tonsils of clinically nontuberculous children.

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139. Coussieu, H.: Migration of a Round Worm into the Ear. Rev. de Laryngologie, d'Otologie et de Rhinologie **39**:358, 1918. Abst. in Archiv. Pediat. **36**:289, 1919.

140. Ransom, B. H.: Ascaris as a Cause of Pulmonary Disease, J. A. M. A. **73**:1210 (Oct. 18) 1919.

141. Kolmer, J. A.: Prenatal Syphilis, with a plea for its Study and Prevention, Am. J. Dis. Child. **19**:344 (May) 1920.

142. Jeans, P. C.: Cerebral Involvement in Hereditary Syphilis, Am. J. Dis. Child. **18**:173 (Sept.) 1919.

143. Austin, R. S.: Bacillus Tuberculosis in the Tonsils of Children Clinically Nontuberculous, Am. J. Dis. Child. **18**:14 (July) 1919.

Opie and Anderson,[144] in an article on the mode of infection with tuberculosis, state that tuberculous meningitis has been followed by recovery, with calcification of those meningeal tubercles which have undergone caseation. The point is emphasized that, in general, the distinction between latent and clinical tuberculosis, which is not infrequently made, has no other basis than the limitations of diagnostic methods, and the tendency of tuberculosis is to proceed to recovery.

The occurrence of a tuberculoma the size of a hickory nut in the midbrain of a child aged 3 years, is reported by Knox.[145] The symptoms were present for three months before death. The child gradually developed bilateral ptosis, partial paralysis of the ocular muscles, and a coarse tremor followed by ataxia. The mass involved corpora quadrigemina and occluded the aqueduct of Sylvius.

An instance of congenital tumor of the brain (telangiectasis) is reported by Spiller.[146]

Amyotomia, etc.—Swanberg and Haynes [147] report a case of mongolism in one of twins.

Starck [148] reports the interesting occurrence of apparent sex incidence in Tay-Sacks amaurotic family idiocy. In a nobleman's family in which the parents were apparently healthy, living in easy circumstances, and without history of Jewish ancestry, the first and fourth child—both girls—had died of the disease at 12 months. The second and third children, boys aged 12 and 10 years, respectively, developed normally and showed no signs of the disease. The fifth child, a girl, was 1 year old when seen by Starck, and seemed likely soon to die.

An unusual number of cases of amyotonia congenita (Oppenheim) are reported by Pearce,[149] the second and third and the fourth and fifth are believed to be the first authentic instances reported of twins suffering from the disease. In the only other instance of this found in the literature, one of the two infants died intrapartum. Krabbe [150] reports six cases in which the disease clinically closely resembled amyotonia congenita, and only later information showed it was a familial disease. He says that according to the original description, amyotonia congenita seems to be a rather benign and not an hereditary disease, and

144. Opie, E. L. and Anderson, H.: First Infection with Tuberculosis by Way of Lungs, Am. Rev. Tuberc. **4**:629, 1920.

145. Knox, J. H. M.: Lesions of the Midbrain, Am. J. Dis. Child. **20**:436 (Nov.) 1920.

146. Spiller, W. G.: Congenital Tumor of Brain (Telangiectasis) and Associated Cerebal Movements, Arch. Neurol, & Psychiat. **2**:50 (July) 1919.

147. Swanberg, H., and Haynes, H. A.: Case of Mongolism in One of Twins, Arch. Neurol. & Psychiat. **1**:717 (June) 1919.

148. Starck: Amaurotic Family Idiocy. Monatschr. f. Kinderh. **18**:139, 1920.

149. Pearce, N. O.: Amyotonia Congenita, Am. J. Dis. Child. **20**:393 (Nov.) 1920.

150. Krabbe, K. H.: Congenital Familial Spinal Muscular Atrophies and Their Relation to Amyotonia Congenita, Brain **43**:166, 1920.

does not seem to leave any defects in adults. In eight of eleven cases which have come to necropsy there have been severe atrophies of the anterior horn cells and muscles. In two of his cases of hereditary congenital muscular atrophy corresponding changes were seen—changes that resemble, on the whole, the changes in Werdnig-Hoffmann's progressive muscular atrophy.

The case described by Holmes [151] conformed clinically in all essential details to the condition known as amyotonia congenita (Oppenheim). The disease, however, was present at birth; there was no lasting amelioration, and the child died of the usual pulmonary complications at the age of 18 weeks. The spinal cord was relatively large for the age of the child; the contour of the cord in cross section was not altered; the anterior roots were diminished in size as compared with the posterior roots. Myelinization was normal, and the cells of Clarke's column were of normal size and well preserved. The large (motor) cells of the anterior horns were strikingly few in number and, in part, replaced by much smaller but apparently healthy looking cells. There was no microscopic evidence of any acute, or chronic, inflammatory process, nor of recent degeneration. There was no gliosis.

In the muscles, areas (bundles) of hypertrophied, but otherwise normal, muscle fibers were intermingled with areas of very small muscle fibers that appeared less fully developed and in appearance resembled most closely embryonic muscle cells of the third month. There was no increase of connective tissue, nor evidence of replacement of muscle by adipose tissue.

The histopathologic findings seemed best explained on the assumption of a delayed or retarded embryonic development affecting certain motor cells of the anterior horns and certain groups of developing muscle cells (fibers).

Pearce's first case is of interest because of the classical symptomatology and the results of the exhaustive metabolism study that was carried out. The following findings and deductions were made: (1) a lowered creatinin excretion, in addition to the excretion of creatin on a low protein diet; (2) a normal uric acid excretion; therefore no nucleoprotein broken down; (3) an increased rest nitrogen, accompanied by an increased neutral sulphur; (4) a normal phosphorus excretion; therefore no bone disintegration, and (5) a lowered chlorid excretion.

From a review of the literature and of their own case, Hueneckens and Bell [152] believe the conclusion justified that amyotonia congenita

151. Holmes, J. B.: Amyotonia Congenita (Oppenheim) Report of a Case With Full Histopathologic Examination, Am. J. Dis. Child. **20**:405 (Dec.) 1920.

152. Hueneckens, E. J. and Bell, E. T.: Infantile Spinal Progressive Muscular Atrophy (Werdnig-Hoffmann); Report of a Case with Necropsy Findings, Am. J. Dis. Child. **20**:496 (Dec.) 1920.

(Oppenheim) and infantile spinal progressive muscular atrophy (Werding-Hoffmann) are extreme types of the same disease—a view presented some years ago by Spiller; also that they are probably both related to the group of myopathies represented by Erb's juvenile form of muscular dystrophy and the hereditary form of Leyden and Moebius.[153]

Haushatter reports three more cases of amyotonia. In his second case the child, up to the age of 4, had remained flabby and inert, keeping any position in which he was placed. He then underwent a rapid transformation; he gradually learned to walk, to carry things to his mouth, to get up from the floor, and to dress himself. At 5 he rapidly learned to talk. At 11 he was in classes with boys of his age, and did not seem their inferior mentally. His body was well proportioned, but there was a diffuse, generalized muscular atrophy. In spite of this, he could walk and run.

The Skin in Measles.—Mallory and Medlar[154] describe in detail the findings in the skin lesions of measles as shown in small pieces of skin removed from thirty-five patients. They consider these lesions as being of infectious origin because of their focal character and lack of uniformity in distribution, and as being due to the causal agent of the disease. They find that the endothelial cells lining the capillaries in the earliest lesions often contain from one to four, rarely more, minute, intensely staining, spherical bodies which vary a little in size. In older lesions the bodies are fewer in number and usually more evident at the periphery of the lesion. Later they disappear entirely. It is suggested that these bodies may be the causal agent; possibly a coccus in various stages of digestion. They react positively to the Gram stain. Study of the nasal, pharyngeal, laryngeal and conjunctional secretions, and of the blood in several preparations by dark field illumination and by culture in various mediums, failed to show any micro-organisms which seemed to bear any etiologic relationship to the disease. Study of the endothelial cells lining the capillaries in other acute lesions examined for purposes of control did not reveal similar spherical bodies.

"The Cambridge," Charles Street at Thirty-Fourth.

153. Haushatter, P.: Amyotonia Congenita, Arch. de méd. d. enf. **23**:133, 1920.

154. Mallory, F. B. and Medlar, E. M.: Skin Lesion in Measles, J. M. Research **41**:327, 1920.

BOOK REVIEW

TYPES OF MENTAL DEFECTIVES. By MARTIN W. BARR, M.D., Chief Physician, Pennsylvania Training School for Feebleminded Children, Elwyn, Pa., and E. F. MALONEY, A.B., Professor of English, Girard College. Price, $3.00. Pp. 179, with 31 plates containing 188 illustrations. Philadelphia: P. Blakiston's Son & Co., 1921.

It is the purpose of this book to facilitate the recognition of various forms of mental deficiencies in children. The possibility of improvement by training is given much consideration. Types of the various degrees of deficiency are given. A series of photographs illustrate the various types. These are either chosen none too well or perhaps reproduced none too well. A brief sketch goes with each photograph. These sketches contain too much of the so-called "human interest stuff." From the standpoint of the physician they could be much improved.

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